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VESTIBULO-OCULAR REFLEX ARC

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THE PROBLEM

The actual anatomic foundations of the concept of the reflex arc were laid by Ramón y Cajal between 1887 and 1892, when he carried out a systematic study of the spinal cord with the method of Golgi. Cajal's findings were soon confirmed by the best neuro-anatomists (van Gehuchten, von Lenhossek, Held, Retzius, von Kölliker and others).[1] A short time later, when Cajal and van Gehuchten developed the concept of axonal polarization, the elementary reflex arc acquired a more general significance; it became the expression of the fundamental plan of structure and function of the nervous system. Figure 1 reproduces the original diagram of Cajal, showing the reflex arc composed of the afferent sensory neuron, the efferent motoneuron and a connecting link that may either be only a collateral from the posterior root or also include one or more intercalated (internuncial) neurons. When the internuncial neurons lie outside the spinal cord, the so-called long-circuiting arcs appear (Fulton[2]); an excellent diagram of Bayliss, indicating the main short and long arcs involved in the production of movements, may be found in Ranson's[3] book. According to these diagrams the nervous system is to be considered as a system of neuron chains involving a varying number of cells superimposed on the elementary two or three neuron reflex arc (fig. 1).

The elementary reflex arc, however, is still only an anatomic idealization of the structure of the nervous system, and thus far no proof exists of its functioning as assumed. Graham Brown[4] came to the conclusion that the fundamental unit in the constitution of the nervous system must be, not the elementary reflex arc, but groups of neurons

From the Central Institute for the Deaf.

1. Ramón y Cajal, S.: Histologie du système nerveux de l'homme et des vertébrés, Paris, A. Maloine, 1909, vol. 1.

2. Fulton: Muscular Contraction and the Reflex Control of Movement, Baltimore, Williams & Wilkins, 1926.

3. Ranson, S. W.: The Anatomy of the Nervous System, ed. 4, Philadelphia, W. B. Saunders Company, 1931, fig. ~~32~~ 33.

4. Brown, T. G.: Die Reflexfunktionen des Zentralnervensystems mit besonderer Berücksichtigung der rhythmischen Tätigkeiten: II, Ergebn. d. Physiol., 1916, vol. 15.

arranged in half-centers and capable of rhythmic activity; of still greater value is the fundamental observation of Herrick and Coghill [5] that in the larval Amblystoma there develops first a highly complicated reflex arc that is able to set up swimming movements, while the so-called elementary arcs develop much later. Working on the vestibular system, I found that after the posterior longitudinal bundle is cut, the labyrinthine reflexes of the eyes can still be elicited; [6] the arc through this pathway is homologous with the elementary spinal arcs. These and many other facts seem to indicate that the so-called elementary spinal arcs do not represent the functional units of the nervous system. That, during the transmission of impulses through the spinal cord, complicated phenomena occur which cannot be explained by means of the customary

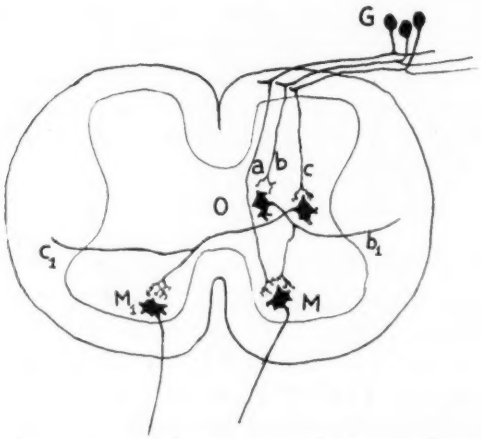

Fig. 1.—The elementary spinal reflex arcs (redrawn after Ramón y Cajal: Les nouvelles idées sur la structure du système nerveux chez l'homme et chez les vertébrés, Paris, C. Reinwald & Cie, 1894). *G* represents the sensory cells of the spinal ganglia; *a*, *b* and *c* are collaterals from the posterior column; *a* ends on motor cells *M* on the same side; *b* ends on an internuncial neuron which originates a fiber (b_1) of the white substance, but first gives off a collateral ending on the motor neuron *M*; *c* ends on an internuncial neuron, which originates a fiber (c_1) of the contralateral white substance, but first gives a collateral ending on the motor neuron M_1.

two-neuron reflex arc has been forcibly stated by Gasser and Graham.[7] On the other hand, it must be kept in mind that even anatomically the

5. Herrick, C. J., and Coghill, G. E.: The Development of Reflex Mechanism in Amblystoma, J. Comp. Neurol. **25**:65, 1915.

6. Lorente de Nó, R.: (*a*) Die Labyrinthreflexe auf die Augenmuskeln nach einseitiger Labyrinthexstirpation nebst einer kurzen Angabe über den Nervenmechanismus der vestibulären Augenbewegungen, Vienna, Urban & Schwarzenberg, 1928; (*b*) Monatschr. f. Ohrenh. **61**:857, 1066, 1152 and 1300, 1927.

7. Gasser, H. S., and Graham: Potentials Produced in the Spinal Cord by Stimulation of Dorsal Roots, Am. J. Physiol. **103**:303, 1932.

elementary arc may not always be considered as being the fundamental factor in the structure of the nerve centers and pathways. For instance, if Cajal's book [1] is carefully read, one finds that in certain cases special cells or fibers are present which establish a new kind of transmission.

A good example is offered by the cerebellar cortex (fig. 2) in which the impulses conducted by the mossy fibers are transmitted first to the granular cells and then to the basket and Purkinje cells. This transmission system (fig. 2, *1*) corresponds to the unidirectional elementary reflex arc; but the parallel fibers also excite the Golgi cells (fig. 2, *2*) which in their turn again excite the granular cells; these impulses go in a direction opposite to that of the former ones, and arrive again at the cell origin of the disturbance. On the other hand,

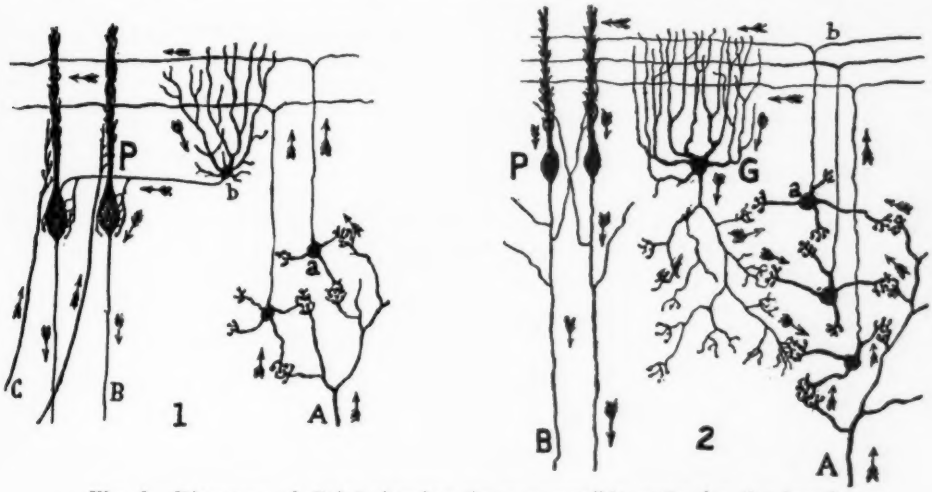

Fig. 2.—Diagrams of Cajal showing the two possible paths for the impulses carried by the mossy fibers to the cerebellar cortex; the arrows indicate the direction of transmission of the impulses. *1* represents the unidirectional arc: *A*, mossy fiber; *B*, axons of Purkinje cells; *C*, climbing fibers; *a*, granule cells; *b* basket cells, and *P*, Purkinje cells. *2* represents the recurrent arc through the Golgi cells (*G*).

the impulses set up by the granule cells arrive at the Purkinje cells twice, once through the parallel fibers (fig. 2, *2*) and then through the basket cells (fig. 2, *1*). Similar examples are found in the cerebral cortex, olfactory bulb, etc.[8] However, the existence of such complicated circuits has been disregarded, undoubtedly because they seemed to be present in the highly complicated organs, such as the cerebral and cerebellar cortex; in the spinal cord, medulla oblongata and midbrain,

8. Ramón y Cajal,[1] 1911, vol. 2. Herrick, C. J.: An Introduction to Neurology, Philadelphia, W. B. Saunders Company, 1927, pp. 111, 220 and 254. Lorente de Nó, R.: La corteza cerebral, Trab. Lab. Inv. biol. Madrid **20**:32, 1922.

where neither cells with short axis-cylinders nor recurrent (centrifugal) fibers had been described, only elementary arcs, such as those in figure 1, could be considered.

During the last twelve years I have been conducting studies on the structure of the nervous system with the purpose of determining whether the general plan of structure is represented by the unidirectional transmission system of figure 1, or rather by circuits similar to those in figure 2. To answer this question it was necessary to investigate, with the help of Golgi's and Cox's methods, the structure of the different nuclei and their connecting pathways and to determine whether or not cells with short axis-cylinders and recurrent collaterals or centrifugal fibers are present. The studies have been conducted on the first segment of the cervical cord, medulla oblongata, cerebellum, midbrain, thalamus and cerebral cortex, and special attention has been

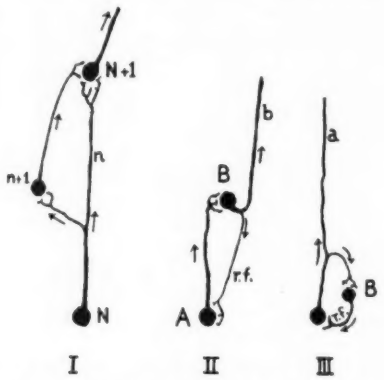

Fig. 3.—Diagrams of the connections within the chains of neurons. *I* represents the law of the plurality of connections; *II* and *III*, the law of reciprocity of connections. The arrows indicate the direction of the transmission of the impulses according to the law of axonal polarization (Cajal); this is also called the law of irreversibility of the synapse (Sherrington).

paid to the trigeminal system, which is homologous with the spinal cord, the acoustic system, which, besides reflex arcs, mainly contains pathways conducting impulses for sensations, the vestibular system and the reticular substance which is composed of internuncial relays. In all systems that have been examined for their presence, cells with short axis-cylinders and recurrent fibers, either collateral or terminal, have been found, so that the following two laws [9] are demonstrated:

1. *Law of Plurality of Connections* (fig. 3, *I*).—If the cells in the spinal or cranial ganglia are called cells of the "first order" and the

9. I first mentioned these two laws in a lecture delivered in the neurologic clinic of the University of Strasbourg, at the invitation of Professor Barré, in February, 1930.

following ones in the transmission system cells of the second, third to . . . nth order, it can be said that each nucleus in the nervous system always receives fibers of at least n and $n + 1$ order, and often of n, $n + 1$ and $n + 2$ order.

This is indicated in diagram *I* of figure 3. Between the nuclei N and $N + 1$ there exist always two kinds of pathways; one pathway n goes direct from N to $N + 1$; the other pathway $n + 1$ encloses an additional neuron; therefore, the impulses set up by the cells of N may arrive at nucleus $N + 1$ twice, once conducted through fibers n and a second time conducted by cells and fibers $n + 1$.

The cells $n + 1$ may be located in the same nucleus as cell $N + 1$ or in a different one. In the first case they are cells with short axis-cylinders, as for instance those of the trigeminal nuclei, of the vestibular (fig. 6) and cochlear nuclei and of the cerebral cortex. In the second case cells $n + 1$ may have long axons; the best examples are the cells of the reticular substance in the medulla oblongata, midbrain and thalamus and the intermediate nucleus of Cajal in the spinal cord, etc. Figure 26 gives a good example.

The plurality of connections has often been mentioned in special cases;[10] for instance, the previously mentioned Bayliss diagram contains numerous pathways between the afferent neurons and the motoneurons. However, there is a fundamental difference between the long-circuiting concept and the concept expressed in law 1; each one of the long-circuiting paths is already a system of neurons of type I (fig. 3); besides, the different paths in the Bayliss or similar diagrams are thought of as having more or less different functions while paths n and $n + 1$ (fig. 3) must be thought of as being used by the impulses at the same time and for the same function.

2. *Law of Reciprocity of Connections.*—If cell complex A sends fibers to cell or cell complex B, B also sends fibers to A, either direct or by means of one internuncial neuron (fig. 3, *II* and *III*).

Cells A and B may lie in the same nucleus (fig. 3, *III*); in this case A is the efferent cell and B is a cell with a short axis-cylinder; in the chain A-B a third neuron may often be intercalated between A and B. Intragriseal A-B chains may be seen in figures 5 and 8.

In other cases, A and B lie in different nuclei (fig. 3, *II*); then the *r.f.* fibers shown in figure 3 are the so-called recurrent or centrifugal fibers. The existence of centrifugal fibers was discovered by Cajal in the retina and olfactory bulb. Later, Cajal and Probst described centrifugal fibers from the cerebral cortex to the thalamus, and recently numerous authors (see literature and discussion in Poljak[11]) have confirmed and completed their descriptions. But the centrifugal fibers were

10. Ramón y Cajal,[1] vol. 2, p. 590; Trab. d. lab. de invest. biol. Univ. de Madrid **1**:125, 1902. He put forward the view that the cells with short axis-cylinders, as in the cortex of the cerebellum, fascia dentata or olfactory bulb, might serve the purpose of reenforcing weak afferent impulses. C. J. Herrick[8] also favors a similar view. This question has been discussed at some length in another paper (Lorente de Nó, R.: Studies on the Structure of the Cerebral Cortex, J. f. Psychol. u. Neurol., to be published).

11. Poljak, S.: The Main Afferent Systems of the Cerebral Cortex in Primates, University of California publications in Anatomy **2**:XIV 370: 1932.

considered as a peculiarity of centers of high order and therefore not as a general trait of structure of the whole nervous system. However, they may be found everywhere if appropriate methods (Golgi's method) are used. One of the systems of centrifugal fibers for the tuberculum acusticum has been described elsewhere;[12] figure 4 reproduces centrifugal fibers for the primary vestibular nuclei. The recurrent fibers from the reticular nuclei for the primary trigeminal nuclei are represented in figure 5 by the axon of cell 4. These three examples referring to the primary nuclei of the fifth and eighth nerves will suffice.

Two exceptions to this law must be mentioned: the sensory spinal ganglia and the motoneurons of the cranial nerves. The sensory ganglia seem not to

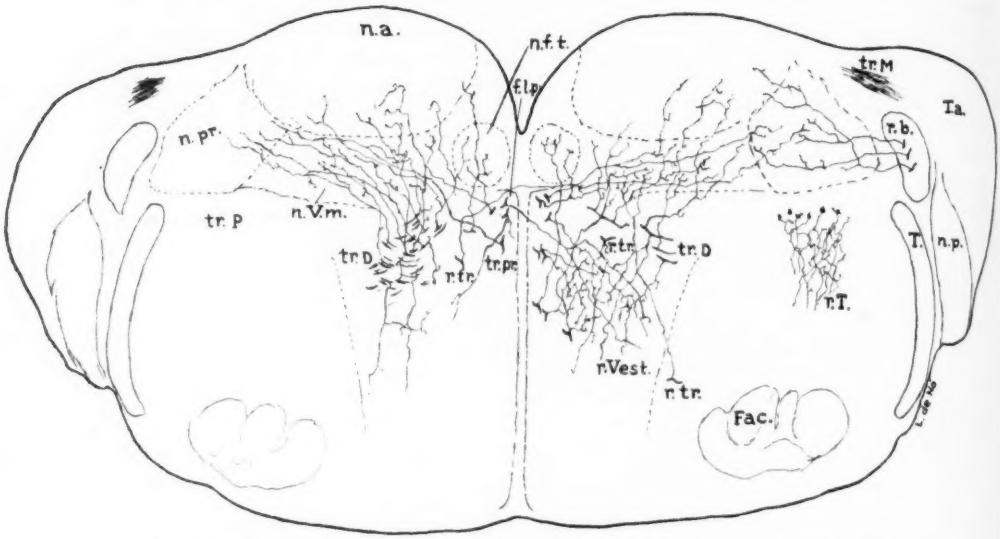

Fig. 4.—Recurrent collaterals (centrifugal fibers) in the vestibular system, stained by Golgi's method. The figure represents a transverse section through the medulla oblongata of a mouse, 7 days old. *n.a.*, nucleus angularis; *n.p.r.*, nucleus proper of the descending vestibular root; *n.v.m.*, nucleus ventromedialis; *n.f.t.*, nucleus funiculi teretis; *Fac.*, nucleus of the facialis; *T.*, descending trigeminal root; *T.a.*, tuberculum acusticum; *n.p.*, nucleus posterior of the cochlear nerve; *r.b.*, restiform body with collaterals for the vestibular nuclei; *tr.M.*, Monakow's striae; *tr.P.*, Probst's tract giving off collaterals for the reticular nuclei of the trigeminal motor system (*r.T.*). The reticular nuclei of the vestibular system (*r.Vest.*) lie between the raphe and the broken lines. The reticular tracts (*r.tr.*), the fasciculus longitudinalis posterior (*f.l.p.*), the predorsal tract (*tr.pr.*) and Deiter's tract (*tr.D.*) give off extremely numerous collaterals for the reticular nuclei; besides, all these tracts give off recurrent collaterals that ascend toward the vestibular nuclei at the floor of the fourth ventricle and end chiefly in the nucleus ventromedialis, the lower part of the nucleus angularis and the lateral part of the nucleus proper. It must be noted that these collaterals constitute many systems with quite different origins and somewhat different endings.

12. Lorente de Nó, R.: (*a*) Anatomy of the Eighth Nerve, Laryngoscope **43**:1 (Jan.) 1933. (*b*) Reflex Reversal and Interaction of Allied Vestibular Reflexes, Am. J. Physiol. **105**:122, 1933.

receive centrifugal fibers; this question, however, cannot be considered as settled. The motoneurons of the spinal cord have been described as having axons with one or several recurrent collaterals ramified in the gray matter, but the motoneurons of the cranial nerves have axons without collaterals (fig. 7). In the last link of the reflex arcs of the medulla oblongata the recurrent fibers (*B-A*) are therefore absent, and it is now of interest to mention that also in the spinal cord two kinds of motoneurons have been found, those having axons with collaterals and those having axons without collaterals.

The general plan of connections in the trigeminal reflex arc can be represented by the diagram in figure 5. *V. s.* is the afferent sensitive fiber; *s.n.* is the sensitive

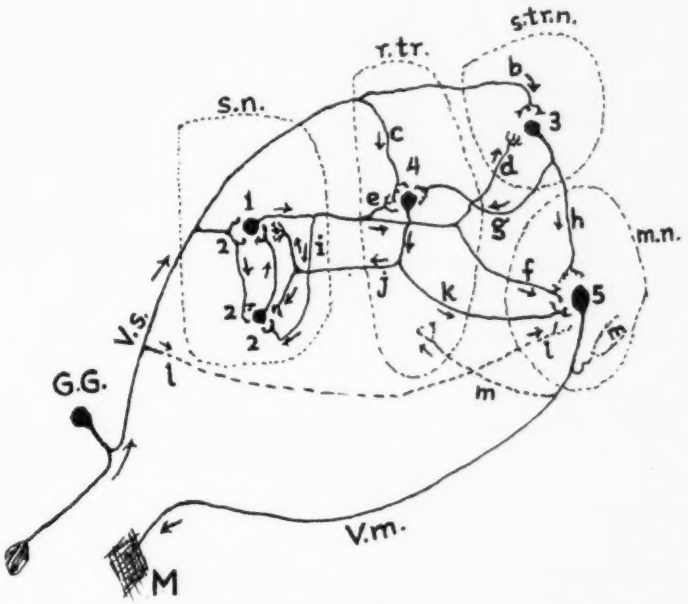

Fig. 5.—Diagram of the connections within the trigeminal reflex arc: *G.G.*, ganglion of Gasser; *V. s.*, afferent trigeminal fiber; *s.n.*, sensory nucleus; *r.tr.*, reticular nuclei of the trigeminal system; *s.tr.n.*, nucleus supratrigeminalis; *m.n.*, motor nucleus; *V. m.*, motor fiber. The arrows indicate the direction of the nervous impulses. A further explanation is given in the text. The actual anatomic relations have been taken in consideration as far as technically possible.

nucleus in the descending root, which contains neurons of two kinds, neurons with a long axis-cylinder (*1*) and neurons with a short axis-cylinder (*2*); *s.tr.n.* is the nucleus supratrigeminalis, which was described for the first time in another paper [13] and has been found again by Castaldi; *r. tr.* is the part of the reticular substance belonging to the trigeminal system (fig. 4, *r.T.*). It is scarcely necessary to mention that *s.n.* and *r.tr.* are composed of several anatomically perfectly delimited nuclei,

13. Lorente de Nó, R.: Contribución al conocimiento del nervio trigémino, Libro en honor de Dn. S. Ramón y Cajal, Madrid, Móya, 1922, vol. 2, p. 13.

and that the pathways indicated in the figure actually are composed of fibers of many different kinds.

The afferent fibers give collaterals in *s.n.* (*2, 2*), *r.tr.* (*c*) and *s.tr.n.* (*b*); the collaterals *s.tr.n.* were described for the first time in the paper mentioned,[13] but no collaterals for the motor nucleus (*m.n.*) were found. The cells with a long axis-cylinder (*1*) of *s.n.* give branches ending on neurons (*2*) (*i*), and branches to *s.tr.n.* (*d*), *r.br.* (*e*) and *m.n.* (*f*); the axons of cells *2* end on cells *1*. The cells (*3*) of *s.tr.n.* give branches to the reticular nuclei (*g*) and to the motor nucleus (*h*); finally, the cells of the reticular nuclei give branches (*j*) to the primary nuclei (*s.n.*) and to the motor nucleus (*k*).

The transmission systems, *V.s.-1-3, V.s.-1-4, 3-4-5, 1-4-5,* are examples of law 1 (plurality of connections within the paths), and the systems *1-2-1, 1-3-4-1* and *1-4-1* are examples of law 2 (reciprocity of connections within the chains of neurons).

If one now attempts to determine on the basis of the diagram of connections which of the many possible paths closes the reflex arc and sets up the motor discharge one is at a loss. Without experimental support no reason can be given for choosing one or the other path, because when the neuron discharges, the impulses travel through all the branches of the axon.

Moreover, if one accepts the theory that the simplest path, *V.s.-1 (f)-5,* sets up the reflex discharge, there would be no use for all the other pathways and nuclei. Since anatomy shows that path *V.s.-1 (f)-5* is only a very small part of the whole system, one has to assume that all the different transmission systems work in intimate collaboration, and therefore neuron *5* discharges only when several or even all the paths are active.

The technical difficulties of experimental work on the trigeminal system, owing to the proximity of the different nuclei, are almost insurmountable; therefore, the attempt has been made to solve the problem in the cerebral cortex and in the vestibular system. In the cerebral cortex it is possible, on the basis of anatomic connections, to assume how the impulses are conducted.[14] In the vestibular system an experimental analysis can easily be accomplished.

But before passing on to the vestibular system it should be mentioned that the diagram of figure 5 will also be valid for some of the spinal reflex arcs if, instead of the nucleus supratrigeminalis, the intermediate nucleus of Cajal is considered. For those motoneurons with axons having recurrent collaterals and for those that have direct connections with the posterior root by means of the so-called sensory motor collaterals, the diagram must be completed with the paths indicated in figure 5 by broken lines (*l, m*).

Since a detailed description of the vestibular system is being published in the *Laryngoscope,* there is need to mention here only the fundamental facts. Cells with a short axis-cylinder of the primary vestibular nuclei have been reproduced in figure 6; figure 7 shows the motoneurons of the abducens nucleus; figure 4 shows recurrent collaterals from the reticular substance and from the fasciculus longitudinalis posterior and tractus predorsalis to the primary vestibular nuclei; figure 8, *I* indicates the plan of structure of the smallest part of the vestibular system that still is able to set up reflexes of the external rectus muscle of the eye, and figure 8, *II* shows the main connections established by the fibers of the posterior longitudinal bundle. Drawings of the actual morphology of the cells were

14. Lorente de Nó, R.: Studies on the Structure of the Cerebral Cortex: I. The Area Entorhinalis, J. f. Psychol. u. Neurol., to be published.

published elsewhere,[15] and there are diagrams of the main pathways of the vestibulo-ocular reflex arc in a later paper.[16]

TECHNIC

The experiments consisted in studying the modifications of the ocular reflexes after lesions in the nervous system destroying one or

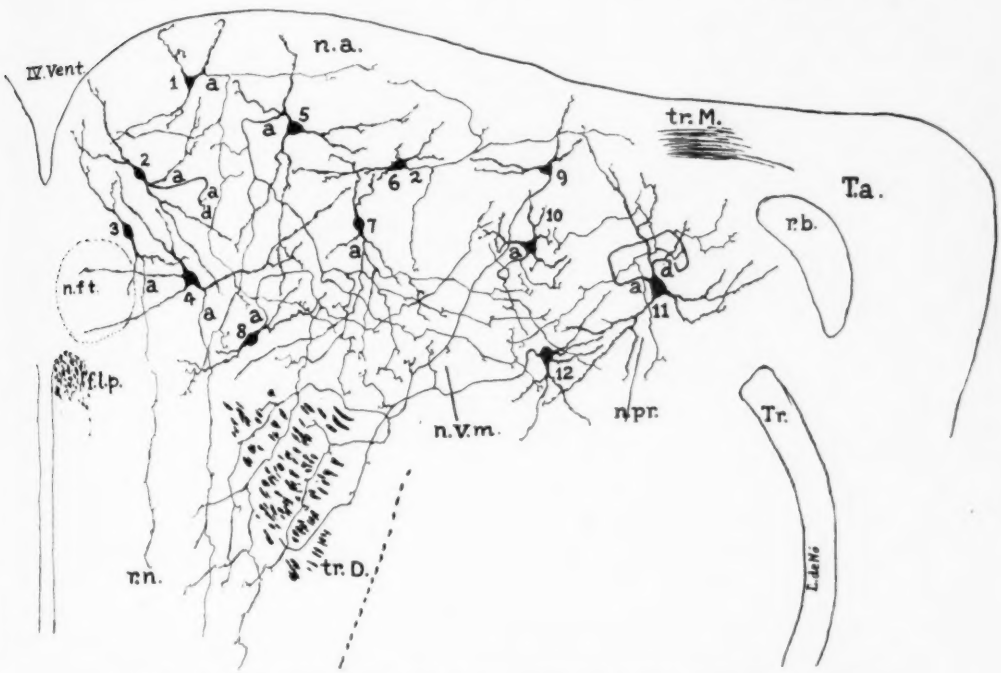

Fig. 6.—Several types of neurons of the vestibular nuclei: *n.pr.*, nucleus proper of the descending root; *n.v.m.*, nucleus ventromedialis; *n.a.*, nucleus angularis; *n.f.t.*, nucleus funiculi teretis; *tr.D.*, Deiters' tract; *f.l.p.*, fasciculus longitudinalis posterior; *r.n.*, reticular nuclei of the vestibular system; *Tr.*, trigeminus; *r.b.*, restiform body; *tr.M.*, tract of Monakow; *T.a.*, tuberculum acusticum; *1* to *12*, several types of cells of the vestibular nuclei, which have either short axons or axons (*a*) ending in the reticular substance. *1* and *2*, cells with short axons; the axon of cell *2* divides in two branches, one ascending (*a*) and the other descending (*d*); *3*, *4* and *8*, cells with axons going to the reticular substance; other cells of this type send their axon to the contralateral reticular nuclei; *5* and *7*, cells with axons ramified chiefly in the nucleus ventromedialis; *6*, a cell sending its axon to the nucleus proper of the descending root; *9*, a cell of the dorsal part of the nucleus proper (pertaining to the maculae) with axon going to the reticular substance; *12*, a cell from the ventromedial nucleus; *10* and *11*, cells with short axis-cylinder of the nucleus proper; the branch *d* of cell *11* constitutes a descending fiber that runs parallel to the vestibular fibers.

15. Lorente de Nó,[6] figs. 42 to 45.

16. Lorente de Nó, R.: Ausgewählte Kapiteln aus der Labyrinthphysiologie, Ergebn. d. Physiol. **32**:79, 1931.

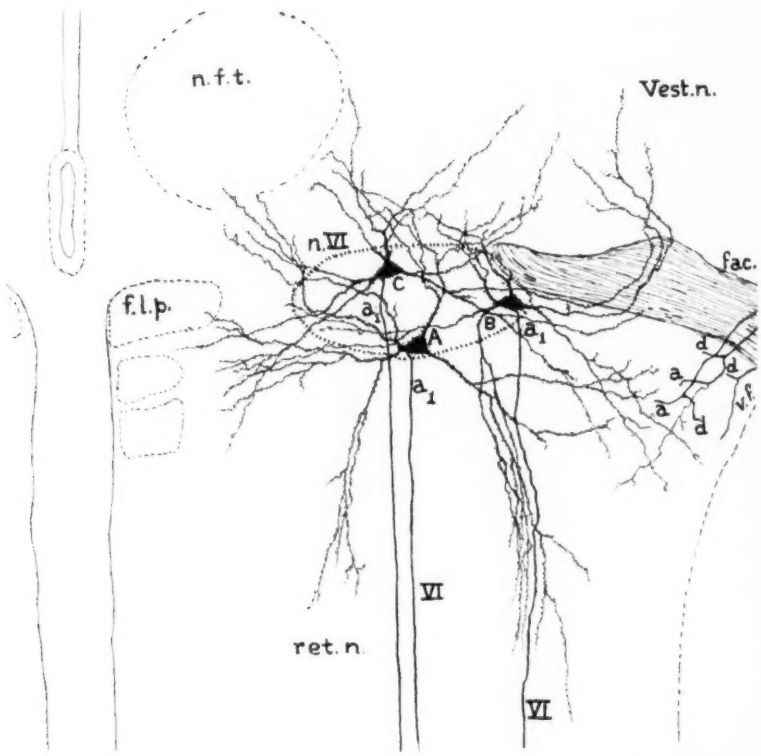

Fig. 7.—Three cells of the abducens nucleus (section from a mouse, stained by the Golgi method): *n.VI*, abducens nucleus; *VI*, fibers of the abducens nerve; *Vest.n.*, vestibular nuclei; *fac.*, facialis nerve; *f.l.p.*, fasciculus longitudinalis posterior; *v.f.*, secondary fibers from the vestibular nuclei, which form in the reticular substance ascending (*a*) and descending pathways (*d*); these fibers are mixed with the fibers of Deiters' tract, which also give numerous collaterals to the reticular substance; *ret.n.*, reticular nuclei of the vestibular system located between the broken lines and the raphe; *n.f.t.*, nucleus funiculi teretis, a part of the reticular system of the vestibular nerve; *A*, *B* and *C*, three cells of the abducens nucleus; a_1, their axons. Note that the body and dendrites of the cells are covered with "thorns," each of them being a synapse. The dendrites go out in three main directions, toward the middle line, upward and downward. The secondary vestibular fibers that constitute the fasciculus longitudinalis posterior give collaterals that end in the interior of the nucleus, but the fibers of the reticular substance establish connections chiefly with the dendrites. It is important that the cells *A*, *B* and *C* have a somewhat different dendritic apparatus; for instance, *C* has its dendrites going chiefly upward, while *B* has long dendrites going downward; therefore, each cell has somewhat different connections. It is important also to note that the connections of the cells are established chiefly beyond the anatomic boundaries of the abducens nuclei; therefore, if only fibers entering the nucleus are considered, the greatest number of synapses are disregarded. The cells have been drawn with the help of a projection apparatus.

several parts of the vestibular system. Previous work [17] revealed the normal characteristics of the reflexes. The method of producing successive lesions proposed in another paper [6] has also been used here.

All experiments were carried out on the rabbit, because in this animal the ocular movements are exclusively or almost exclusively of vestibular or allied origin (neck reflexes). The operations were performed under ether anesthesia; but since the vestibular reflexes disappear during the narcosis, at the beginning of the experiment the cerebral cortex was extirpated to permit the study of the reflexes after total recovery from the narcosis.

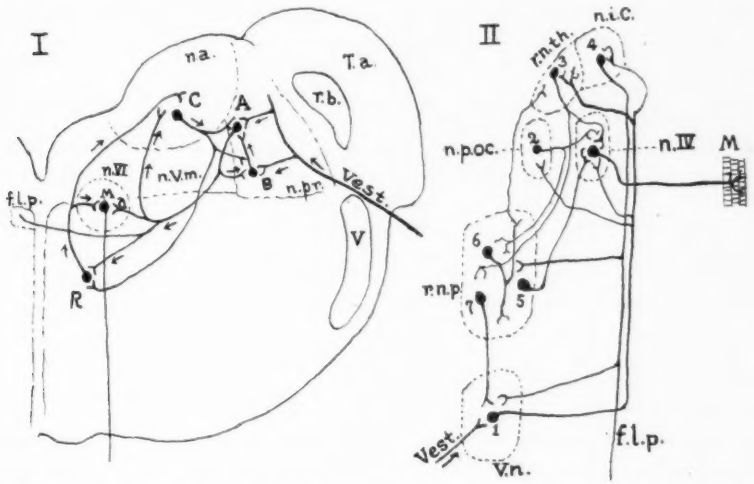

Fig. 8.—*I*, diagram showing the connections of the neurons in the smallest part of the medulla oblongata, which still is sufficient to set up the slow phase of the nystagmus in the external rectus muscle. Each cell of the drawing actually represents a group of neurons having similar connections; and each pathway is either a single fiber or a chain of neurons with the same origin and end (fig. 3, *I*.) *Vest.* is the vestibular afferent fiber; *A*, a cell with a long axis-cylinder; *B*, a cell with a short axis-cylinder; *A* gives branches ending on *B*, and afterward establishes connections with the motor nucleus of the abducens and with the reticular substance; *B* gives off branches ending on *A*; R gives off branches for the abducens and angularis nuclei; *C* gives off branches ending in the reticular substance and on cells *A* and *B*; *n.v.m.*, nucleus ventromedialis; *n.a.*, nucleus angularis; *n.VI*, abducens nucleus; *R*, reticular nuclei (a further explanation is given in the text). *II*, diagram of the main connections of the posterior longitudinal bundle; *Vest.*, vestibular afferent fiber; *V. n.*, vestibular nuclei; *f.l.p.*, posterior longitudinal bundle; *n.p.oc.*, nucleus para-ocularis, one hitherto apparently undescribed part of the vestibular system; *r.n.p.*, reticular nuclei in the pons; *r.n.th.*, reticular nuclei in the thalamus; *n.i.C.*, nucleus interstitialis of Cajal. The pathways *V. n.-n.IV*; *v.n.-n.p.o.c.*, *n.III*; *V. n.-r.n.th.-n.IV*; *V. n.-n.i.C.-r.n.p.-n.IV*, *etc.*, are examples of law I; and the pathways *V. n.-n.i.C.-V. n.*; *V. n.-n.i.C.-r.n.p.-V. n.*; *V. n.-r.n.th.-r.n.p.-v.n.*, etc., are examples of law II.

17. Lorente de Nó, R.: Untersuchungen über die Anatomie und Physiologie des Nervus octavus, Trab. d. lab. de invest. biol. Univ. de Madrid **25**:159, 1927.

The reflex reactions of the ocular muscles were recorded by means of Bartel's[18] technic, which consists in connecting the individual ocular muscles with isotonic levers. In previous experiments only two muscles at a time were used for recording, but in the actual series the reflexes of the six muscles were recorded simultaneously on a drum 18 inches high. The use of isotonic levers offers disadvantages, but has the advantage that records of long lasting reflexes can be obtained. Isometric recording is necessary when the tensions developed by the muscles or the time relations of the reflexes have to be accurately measured, but in this study the tracings were obtained only for the purpose of having a permanent record of the existence and direction of the nystagmus and of the tonic contractions; for this purpose isotonic recording gives perfectly reliable results.

The excitation of the labyrinth was originally produced by means of rotation, but for certain purposes it was found more advisable to use stimulation of the individual canals. This was done by touching the bony canals with a cold or warm needle. The needles are 1 mm. in diameter, and the point is spherically rounded so that contact with the bone occupies practically less than 0.25 sq. mm. The hot needle is heated to a constant temperature (60 C.) with a resistance coil. The cold needle is kept at the desired temperature by means of a double cannula through which water at 10 C. circulates. Previous experiments demonstrated that only the canal which is touched with the needle is stimulated. A still better proof was found in the actual series of experiments; when one canal, as a consequence of lesions in the vestibular nuclei, becomes ineffective, no reflex reaction appears even if the contact of the needle lasts for several minutes, while if one of the active canals is touched a reaction promptly arises.

The reflexes following caloric stimulation of the semicircular canals have been described in detail elsewhere.[19] When the needle is brought in contact with the canal, after a short latent period, a nystagmus arises which increases in intensity to a certain maximum, dependent on the temperature of the needle, and remains constant as long as the needle rests on the canal. When the needle is taken away the nystagmus remains for a certain time (after-discharge). In the case of rotations the after-discharge is of course almost exclusively of central origin, because the physical processes in the labyrinth last for only a small part of a second,[20] but in the case of caloric excitation the after-discharge is composed of two parts: (*a*) the time necessary for the bony canal to recover the body temperature and (*b*) the central after-discharge.

It would be justified to take as duration of part (*a*) the duration of the shortest observed after-discharge, but in order to avoid complications no reference will be made in this paper to the duration of the after-discharge except when it was determined before the operation or when it became so long that no doubt about its central origin arose.

The lesions in the vestibular nuclei were made after extirpation of the cerebellar vermis; this eliminated the possibility of accidental lesions during the successive operations. When the extirpation of the vermis is totally symmetrical no spon-

18. Bartels, M.: Ueber Regulierung der Augenstellung durch den Ohrapparat, Arch. f. Ophth. **78**:129, 1911.

19. Lorente de Nó (footnotes 16 and 17).

20. Schmaltz, G.: The Physical Phenomena Occurring in the Semicircular Canals During Rotary and Thermic Stimulation, Proc. Roy. Soc. (Sect. Otol.) **25**:1, 1931. Rohrer and Masuda: Physikalische Vorgänge in Bogengangsapparat und Statolithenapparat, Handb. d. norm. u. path. Physiol. **11**:985, 1926. Lorente de Nó,[6a] chap. 10 (mathematical calculations and experimental results).

taneous nystagmus appears, but if it is somewhat asymmetrical a weak spontaneous nystagmus arises, which adds itself to the vestibular reflexes, but without masking them. At the beginning of this series of experiments the vestibular reflexes were always recorded after extirpation of the cerebellum, prior to any lesion of the vestibular nuclei, but as it was always found that every canal was effective, in the last experiments the first lesion in the floor of the fourth ventricle was made immediately after the extirpation of the vermis.

For histologic analysis the brains were fixed in formaldehyde, cut in series and stained with Weigert's iron hematoxylin.

To depict the lesions, drawings have been made with the help of the projection apparatus (figs. 9, 11, 15, 17, 19, 21 and 24).

The scheme of each experiment was: (1) preparation of one or both labyrinths and of the muscles in one or both eyes; extirpation of the cerebral cortex; extirpation of the cerebellar vermis; (2) ocular reflexes recorded after total recovery from the narcosis; (3) first lesion in the floor of the fourth ventricle; (4) ocular reflexes

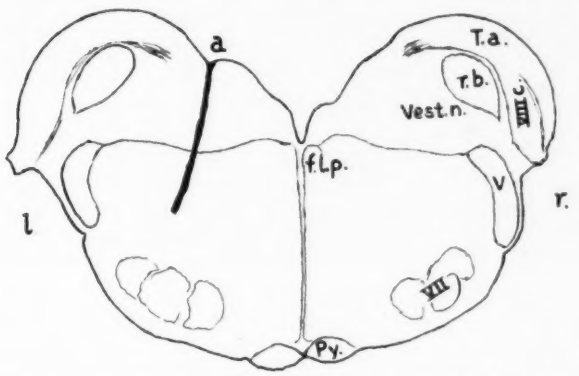

Fig. 9 (rabbit 1).—Transverse section through the medulla at the level of the facialis nucelus. In this and the following drawings the abbreviations are as follows: *A.S.*, acqueductus sylvii; *Br.c.*, brachium conjunctivum; *Br.p.*, brachium pontis; *C.p.*, colliculus posterior; *Cer.*, cerebellum; *f.l.p.*, fasciculus longitudinalis posterior; *G.v.*, ganglion ventrale; *L.l.*, lemniscus lateralis; *M.str.*, striae monakowi; *O.s.*, superior olive; *P.*, pons; *Py.*, pyramid; *q.f.*, quadrangularis field; *r.b.*, restiform body; *T.a.*, tuberculum acusticum; *T.n.*, trapezoid nucleus; *V*, descending root of the fifth nerve; *VI*, abducens; *VII*, facialis; *V. s.*, sensory trigeminus; *V. m.*, motor trigeminus; *Vest.*, vestibularis; *Vest.n.*, vestibular nuclei; *a*, *b* and *c*, lesions.

again recorded; (5) second lesion in the floor of the fourth ventricle; (6) ocular reflexes again recorded, etc.

As already mentioned, in the last experiments step 2 was omitted. None of the facts described in this paper can be attributed to shock following the operations because, first, the rabbit does not show shock [21] and, second, I shall refer exclusively

21. Magnus (Körperstellung, Berlin, Julius Springer, 1924) mentioned that in a case of decerebration in a rabbit, although the section passed through the oculomotor nucleus, the eyes still showed nystagmus; this indicates that in the rabbit there is no danger of shock. A similar case is shown in figures 11, 12 and 13 of the present paper.

to positive findings or to the absence of reflexes in some muscles only when the existence of reflexes of other muscles excludes the existence of shock.

Over thirty experiments have been carried out in this series, of which only ten that are typical and may be reproduced easily will be mentioned here; the others are more or less a repetition, intended to corroborate the results obtained. Since I am dealing now only with the vestibulo-ocular reflex arc, the modifications of the other vestibular reflexes will not be described; besides, this allows the exposition to be short.

EXPERIMENTAL RESULTS

EXPERIMENT 1.—*Jan. 15, 1932. Right eye.*

First Operation.—Extirpation of the left labyrinth was performed.

A spontaneous nystagmus having the characteristics described elsewhere[6] appeared. Excitations of the right labyrinth brought forth the customary reactions.

Second Operation.—Extirpation of the cerebellar vermis and two punctures similar to the lateral ones in figure 24, *II*, *a*, *b*, but smaller, were done. The homolateral pathways from the oral part of the vestibular nuclei (tractus vestibulomesencephalicus) were cut.

The spontaneous nystagmus ceased; only the superior rectus showed a very small one (fig. 10, *II*). The excitations of the right labyrinth gave rise to reflex reactions of only the superior and the internal recti.

The superior rectus answered only with an increase or decrease of its spontaneous nystagmus (fig. 10, *II*), but the internal rectus showed nystagmus in both directions. Although some of the muscles did not respond to labyrinthine stimuli, they responded readily to excitations of the conjunctiva (corneal reflex), giving curves extremely similar to those in figure 12.

Third Operation.—A longitudinal lesion on the left side of the medulla (fig. 9, *a*) that began at the level of the entrance of the vestibularis nerve and, always at the same distance from the middle line, reached the level of the calamus scriptorius. Through this operation the greater part of the primary nuclei of the already extirpated labyrinth were disconnected; i. e., the pathways connecting them with other structures in the medulla were cut.

The spontaneous nystagmus disappeared totally; of the six muscles only the external rectus responded to excitations of the right labyrinth. The reaction to the current from the ampulla in the horizontal canal was normal in direction (nystagmus with quick relaxations, fig. 10, *III*, *5*), but the reaction to the current toward the ampulla was abnormal because it consisted of the same nystagmus (fig. 10, *III*, *6* and *7*). The abnormal reaction was accompanied by strong movements of the body, which appeared in the tracings as irregular oscillations of the base line (fig. 10, *III*, *c*).

Comment.—After the second operation, several muscles, including the external rectus, were unable to respond to labyrinthine impulses, although they responded readily to trigeminal ones (corneal reflex). There can be no doubt that the inhibition of the labyrinth reflexes was due to an inhibition not of the motoneurons, but of some other neurons further upstream. The inhibition of the external rectus was relieved through the third operation, and the muscle again reacted (fig. 10, *III*).

The available information is not sufficient to determine which neurons were the site of the inhibition, but the following considerations lead to important conclusions: (*a*) The stimuli setting up the reflex reactions were always produced in the right vestibular nuclei. Between these nuclei and the abducens nucleus there were unbroken pathways (fig. 8, *I*, *a*) which could not be the site of inhibition. Since

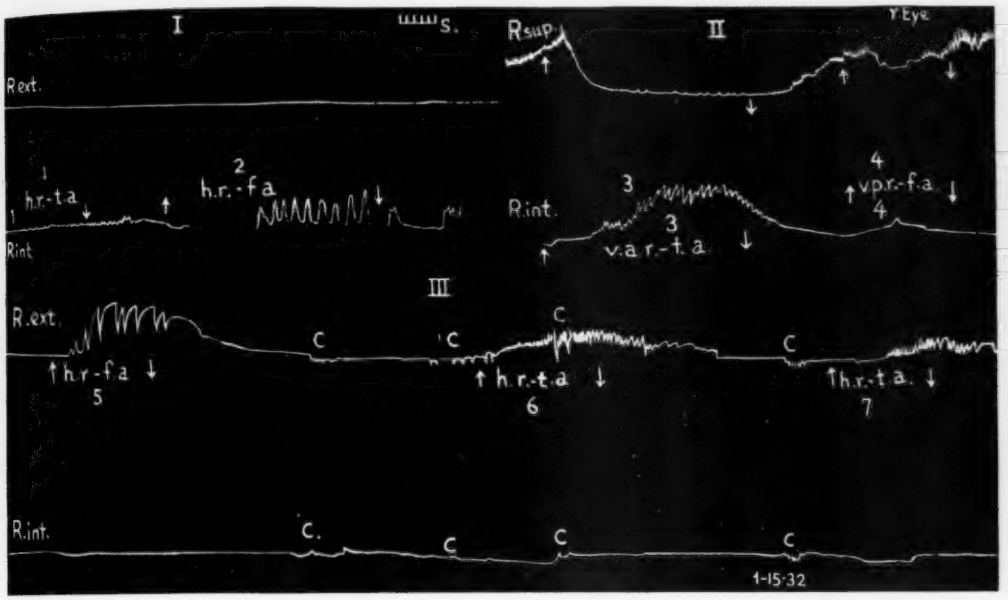

Fig. 10 (rabbit 1).—Some of the reflex reactions of the muscles of the right eye: *I* and *II*, after the second operation; *III*, after the third operation. The tracings are to be read from left to right. Rising of the tracing indicates contraction; the shortening of the muscle appears in the original tracing magnified six times. The beginning of the stimulation of the canals is indicated with upward pointing arrows; the end, with downward pointing arrows. *S* represents the time in seconds; *h.*, horizontal; *v.a.*, vertical anterior canals; *v.p.*, vertical posterior canals; *r.*, right; *l.*, left; *t.a.*, current toward the ampulla; *f.a.*, current from the ampulla. Thus *h.r.f.a.* indicates current from the ampulla in the right horizontal canal, etc. *I*, excitations of the horizontal canal of the right labyrinth; *1*, current toward the ampulla, *2*, current from the ampulla; *R. int.* shows nystagmus of the expected direction; *R. ext.* shows no reaction. *II*, excitations of the vertical canals; *R. sup.*, spontaneous nystagmus with quick relaxations which becomes of much less intensity when either a current toward the ampulla in the vertical anterior canal (*3*) or a current from the ampulla in the vertical posterior canal (*4*) is produced. *R. int.* shows in the first case (*3*) a nystagmus with quick relaxations; in the second case (*4*), defective nystagmus. *III*, excitations of the horizontal canal of the right labyrinth: *5*, current from the ampulla; *R. ext.* shows nystagmus of the expected direction; *6* and *7*, currents toward the ampulla. *R. ext.* shows nystagmus of the same direction as in *5*, i. e., nystagmus of reversed direction. The rabbit moves and the slight displacements of the head appear in the tracings of both muscles (*C*). *R. int.* does not show any labyrinthine reflex, but to excitations of the conjunctiva it responds exactly in the same way as *R. ext.*

the motoneurons were not inhibited, it follows that only one of two alternative explanations is possible. Either the inhibited neurons were those of the primary vestibular nuclei, in which case no impulses were sent to the abducens, or the inhibited neurons were of reticular nuclei, in which case one would have to believe that the fasciculus longitudinalis posterior impulses need reenforcement by reticular impulses to set up motor discharges. This second alternative is favored by other experiments (recorded later).

(*b*) The reactions of the internal rectus after the second operation were still of normal direction, whereas after the third operation the external rectus showed abnormal reactions. Since the primary vestibular nuclei on the right side were not damaged in any way and there is not an unbroken pathway between the primary nuclei on both sides, it is evident that the change in character of the reaction must have been due to abnormal activity of the reticular relays, which as a consequence of the longitudinal section no longer were under the influence of the left primary nuclei.

This fact proves that the reticular nuclei constitute an important part of the reflex arc, and that their impulses, conducted to the primary nuclei through recurrent fibers, are necessary for the production of normal reflexes. This conclusion was already reached after similar experiments in which the excitation of the labyrinth was produced by means of rotation.[22] On the other hand, the doubt arises whether the unbroken pathways alone are able to close the reflex arc and set up motor discharges.

EXPERIMENT 2.—*Feb. 11, 1932. Right eye, left and right labyrinth.*

First Operation.—Extirpation of the cerebellar vermis was done.

Second Operation.—A transverse lesion was made in the floor of the fourth ventricle cutting the fasciculus longitudinalis posterior and the tractus vestibulomesencephalicus on both sides; the lesion was very similar to the transverse lesion (*a-b*) in figure 24, *I*.

No spontaneous nystagmus occurred; the labyrinthine reflexes were apparently the same as after the first operation.

Comment.—This experiment proves beyond doubt that the complicated vestibular arcs closed through the reticular substance are able to set up reflex reactions extremely similar to those produced by activity of the whole system. That all the vestibular reflexes on the ocular muscles (nystagmus, tonic reflexes and third reaction) may still be elicited after total section of the long pathway (fasciculus longitudinalis posterior and tractus vestibulomesencephalicus) was demonstrated in 1927.[6] In these experiments the reflexes were produced by means of rotation. Spiegel also found nystagmus after section of the fasciculus longitudinalis posterior,[23] and Blohmke [24] found that the nystagmus produced by faradic stimulation of the thalamus also takes place after the fasciculus longitudinalis posterior has been cut.

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22. Lorente de Nó (footnotes 6 and 16).

23. Spiegel, E.: Experimental Studien am Nervensystem: XV. Der Mechanismus der labyrinthären Nystagmus, Ztschr. f. Hals-, Nasen- u. Ohrenh. **25**:200, 1929.

24. Blohmke, A.: Ueber den durch elektrischen Reizung des Hirnstammes ausgelösten Nystagmus beim Kaninchen, Ztschr. f. Hals-, Nasen- u. Ohrenh. **24**:520, 1929.

EXPERIMENT 3.—*Jan. 25, 1932. Right eye, right and left labyrinth.*

First Operation.—Extirpation of the vermis of the cerebellum and longitudinal section of the left side of the medulla (fig. 11, *I*) were done. The section, which was lateral to the left fasciculus longitudinalis posterior, began at the level of the caudal end of the abducens nucleus and proceeded downward, always at the same distance from the middle line, to the level of the dorsal nucleus of the eleventh nerve.

No spontaneous nystagmus occurred; only two muscles (superior and external recti) reacted to labyrinthine stimuli, but all of them showed the corneal reflex, apparently in normal fashion (fig. 12).

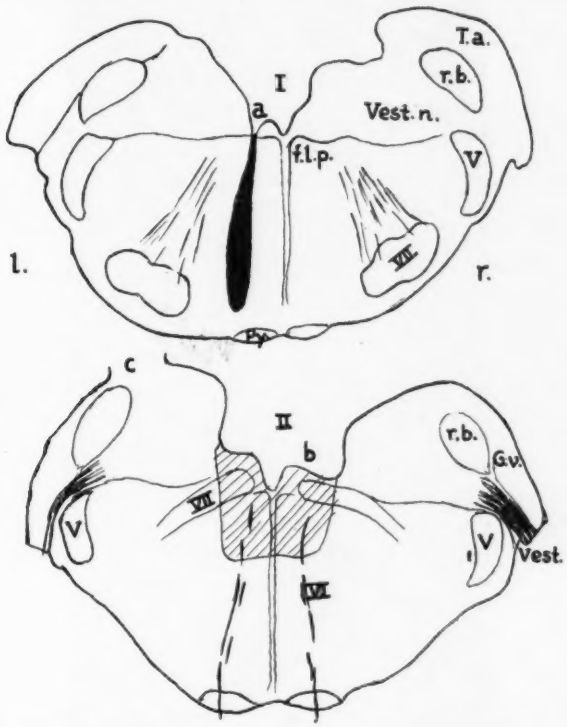

Fig. 11 (rabbit 3).—Transverse sections through the medulla. *I*, section at the level of the facialis nucleus, somewhat caudal to the vestibularis entrance and abducens nucleus: *a*, oral extremity of the longitudinal lesion. *II*, section immediately oral to the facialis knee; *b*, projection of the transverse lesion on this section; a few of the most oral root fibers of the abducens nerve also are cut; however, the rest of the abducens nucleus was able to set up motor contractions (absence of operative shock).

Second Operation.—A transverse lesion was made in the floor of the fourth ventricle cutting the fasciculus longitudinalis posterior on both sides and also a few of the dorsal reticular paths (fig. 11, *II*).

The reflexes were the same as after the first operation. The superior rectus showed nystagmus in the expected direction (fig. 13). The external rectus, which now had a very small spontaneous nystagmus with quick relaxations, showed an

incomplete reaction; when the labyrinthine stimulus had to set up a nystagmus in the same direction as the spontaneous one, this added itself to the strong slow contraction (fig. 14, *2* and *4*); but when the labyrinthine stimulus had to set up the opposite nystagmus, the spontaneous one was inhibited (fig. 14, *1* and *3*); only the current toward the ampulla in the right vertical anterior canal was able to set up a nystagmus with quick contractions in the external rectus (fig. 13, *3*).

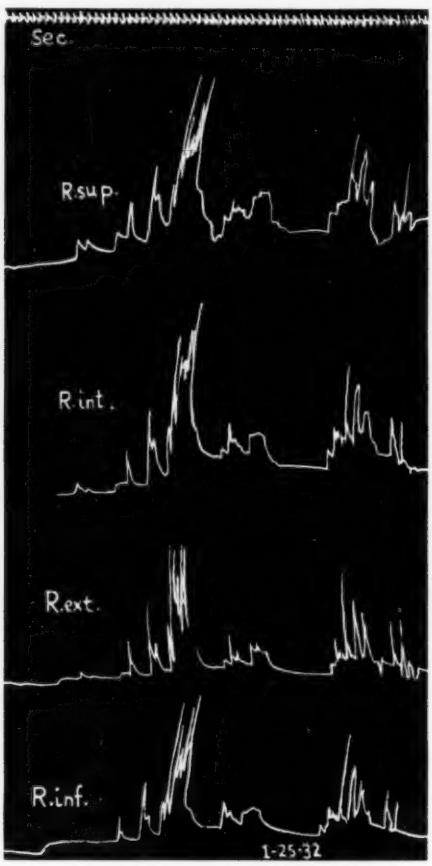

Fig. 12 (rabbit 3).—Corneal reflex of the four rectus muscles of the right eye after the first lesion (fig. 11, *1*, *a*). Note that all the muscles show the reflex in apparently the same way.

Comment.—The first thing to attract attention is that in spite of the asymmetry of the longitudinal section (figs. 11, *1*) both horizontal canals set up exactly the same reflex reactions (fig. 14). The section was on the left side; therefore, the right vestibular nuclei had their long pathways intact, whereas the left ones had lost the crossed paths arising from the posterior two-thirds. To explain the symmetry of the reflexes in spite of the asymmetry of the lesion, it could be thought that the horizontal semicircular canals are represented only in the oral third of the vestibular nuclei, which in this experiment had its pathways intact, but

in view of the anatomy of the vestibular nuclei this assumption is impossible, because the fibers of the semicircular canals are distributed in the whole length of the nuclei at the floor of the fourth ventricle;[12b] besides, it would contradict the results of other experiments (experiment 7).

The only explanation seems to be that the nuclei of both labyrinths influence each other and also act on the motor nuclei by means of some internuncial relay (reticular substance), and that the reflex reactions are determined in agreement with the functional state of this relay.

That the superior rectus responded to excitations of the labyrinths in the same way after the first and second operation shows, in agreement with experiment 2, that the labyrinth reflexes are much alike when the posterior longitudinal bundle is intact and when it has been totally cut. This raises the question as to the rôle played by the fasciculus longitudinalis posterior impulses, and the first problem

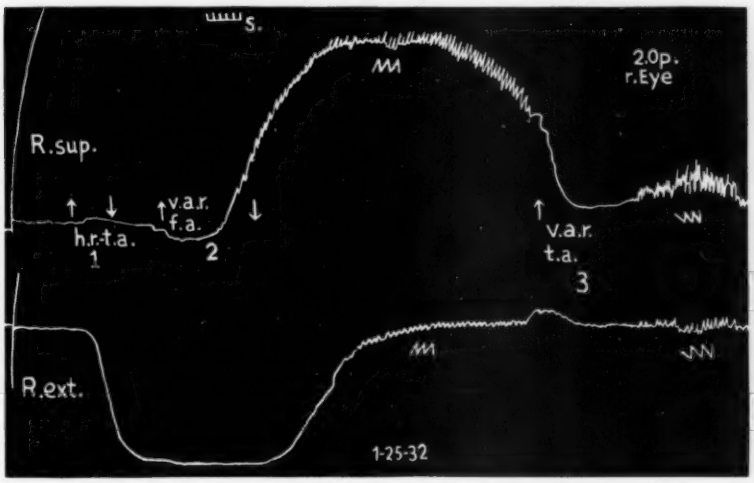

Fig. 13 (rabbit 3).—Some of the reactions of the muscles of the right eye after the second operation: *1*, current toward the ampulla in the right horizontal canal; *R. ext.* shows an extensive relaxation; *2*, current from the ampulla in the right vertical anterior canal; *R. sup.*, after a long latence time, shows a strong nystagmus with quick relaxations; *R. ext.*, after a still longer latence time, contracts and shows the spontaneous nystagmus with quick relaxations; *3*, current toward the ampulla in the right vertical anterior canal; both muscles reverse the direction of their nystagmus and show nystagmus with quick relaxations.

is whether these impulses alone are able to set up motor discharges. The problem has not yet received a definitive answer.

Spiegel[23] tried to destroy the substantia reticularis without damaging the posterior longitudinal bundle, and concluded that this pathway in the absence of the reticular substance is able to close the reflex arc and set up motor discharges. But his experiments, in spite of the excellent technic used, are not conclusive, because the substantia reticularis was only partially damaged and pathways were left intact which, in the absence of the posterior longitudinal bundle, might carry to the motor nuclei impulses bringing forth nystagmus. For instance, in figure 2 of Spiegel's paper the left substantia reticularis is very little damaged, and its

pathways are normal; the lesion in figure 3 is more extensive, but a good deal of the reticularis substance and pathways are still undamaged.

In numerous experiments the attempt has been made to destroy the substantia reticularis without damaging the posterior longitudinal bundle. It has been found more advisable to work through the upper way, i. e., on the floor of the fourth ventricle, because if the lesions are made through the inferior surface of the pons the destruction of the reticular substance is never complete. The experiments have always given conflicting results (compare experiments 4 and 5).

EXPERIMENT 4.—*Jan. 8, 1932. Right eye, both labyrinths.*

First Operation.—Extirpation of the cerebellar vermis was done and a longitudinal section was made in the middle line of the medulla (fig. 15, *II*), beginning a little caudal to the posterior end of the abducens nucleus and reaching the calamus scriptorius.

Every canal was effective and set up nystagmus in the expected direction.

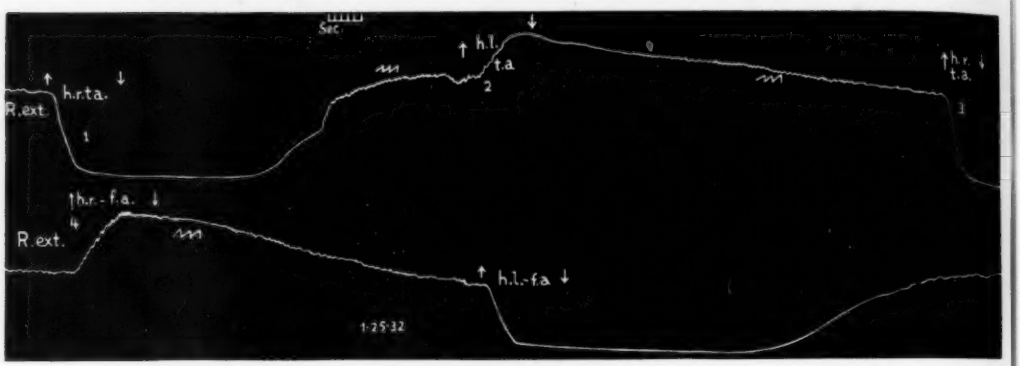

Fig. 14 (rabbit 3).—Reactions of the external rectus muscle to the excitations of the horizontal canals after the second operation. Note that on the one hand, the currents from the ampulla in the right horizontal canal (*4*) and toward the ampulla in the left one (*2*) and, on the other hand, the currents toward the ampulla in the right canal (*1* and *3*) or from the ampulla in the left one (*5*) produce the same reactions in spite of the asymmetry of the longitudinal lesions in the medulla (fig. 11, *I*).

Second Operation.—Two transverse lesions were made in the floor of the fourth ventricle in the pons, which destroyed some of the dorsal reticular paths and the left posterior longitudinal bundle, but left intact the right one (fig. 15, *I*).

All the muscles but the external rectus ceased to respond to labyrinthine stimuli, even to the strongest ones. The external rectus showed only a pseudopostural reflex, which had the direction of the slow phase of the nystagmus. The currents toward the ampulla in the left and toward the ampulla in the right horizontal canal were apparently ineffective, but if pitted against the effective ones prevented the appearance of the expected reaction; i. e., they were unable to set up an actual relaxation of the muscle, but prevented its contraction.

The reactions set up by currents toward the ampulla in the right or from the ampulla in the left horizontal canal (fig. 16) offer a noteworthy property. As a rule, when the needle is applied against the bony canal the intensity of the nystagmus or of the slow component in cases in which the quick one is missing

increases (central summation of the impulses) as long as the needle rests on the canal, or else after having attained a maximum remains with constant intensity until the needle is withdrawn and the bony canal is allowed to recover the body temperature. But in this case, although the ascent of contraction was as rapid as it generally is, afterward, the temperature remaining the same, the muscle progressively relaxed until the reaction disappeared almost totally; but if the excitation was interrupted at any point of the plateau a long after-discharge followed, apparently of normal duration; therefore, it was not the mechanism setting up

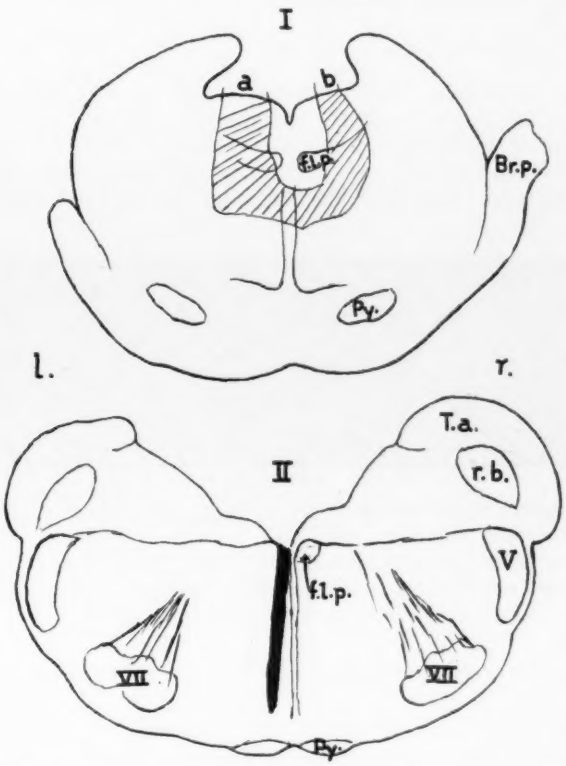

Fig. 15 (rabbit 4).—Tranverse sections through the pons and medulla: *I*, transverse lesion in pons; *II*, oral extremity of the longitudinal lesion in the medulla.

the after-discharge which failed but the nuclei of the vestibularis that became adapted to the peripheral stimulus.

Comment.—The right posterior longitudinal bundle was not able to close the physiologic reflex arc and set up contractions of the muscles innervated by the oculomotor nerve. The impulses which after the first operation were able to set up nystagmus must have been conducted through the reticular paths destroyed in the second operation (fig. 75, *I*).

A new phenomenon appeared in this experiment; the nystagmus, as a consequence of a lesion outside the primary vestibular nuclei, was converted into a pseudopostural reflex; i. e., the quick phase of the nystagmus was suppressed. I

shall discuss this phenomenon later. On the other hand, the course of the reaction had become abnormal, in spite of the fact that the primary vestibular nuclei on the right side were not damaged. This again has no other explanation than that the lesion in the pons had cut some descending (centrifugal) fibers, the activity of which was necessary for the normal function of the primary nuclei. This was demonstrated in other experiments [22] in which it was found that even the oral extremity of the reticular substance in the midbrain has an influence on the activity of the primary vestibular nuclei. Later, Blohmke [25] found that faradic stimulation of the reticular substance in the oral extremity of the midbrain sets up a nystagmus with a long after-discharge.

EXPERIMENT 5.—*May 2, 1932. Both horizontal canals; rectus muscles of the left eye.*

Operation.—Extirpation of the cerebellar vermis was done, and three sections were made in the midbrain with the intention of cutting the reticular pathways without damaging the fasciculus longitudinalis posterior (fig. 17). Both fasciculi were intact and many of the reticularis pathways were cut several times, but some of them immediately underneath the fasciculus longitudinalis posterior and in

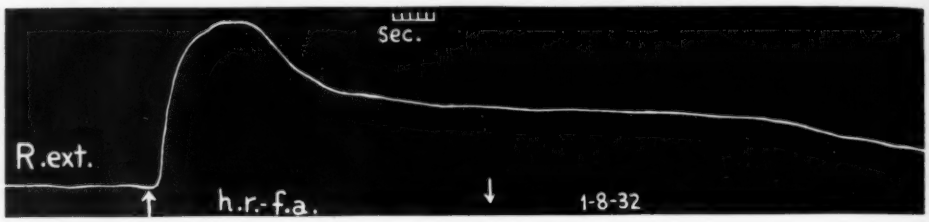

Fig. 16 (rabbit 4).—Reaction of the external rectus to a current from the ampulla in the right horizontal canal after the second operation. Note that the muscle relaxes while the excitation of the canal remains unchanged and that after cessation of the excitation the muscle remains contracted (after discharge), apparently as long as or even longer than it generally does.

the quadrangularis field were still intact. Also the ventrolateral ones remained undamaged.

A spontaneous nystagmus appeared. The external and inferior recti had quick contractions; the internal and superior recti, quick relaxations. The horizontal canals set up the expected reflex reactions, but the after-reaction was enormously long. In this rabbit the duration of the after-reaction had been determined when the nervous system was intact, and therefore it is possible to state that after the operation it was three or four times longer. Figure 18 contains some reactions of the horizontal muscles. The current toward the ampulla in the left horizontal canal set up the expected nystagmus, but it was of enormously long duration; in *2* a current from the ampulla was able to produce only a descent of the plateau after-discharge; in *3* the same excitation was already able to reverse the existing after-reaction, but the resultant nystagmus was weak. Half an hour afterward,

25. Blohmke, A.: Ueber den durch elektrische Reizung des Hirnstammes auslösbaren Nystagmus beim Kaninchen, Ztschr. f. Hals-, Nasen- u. Ohrenh. **23**:213, 1929.

the current toward the ampulla in the right horizontal canal also produced a nystagmus of the direction of that in *3* but this time of the same strength as in *1*.

Comment.—The effect of the operation was a lengthening of the after-discharge and a modification of the character of the nystagmus, which in spite of very strong slow phases, showed only very few quick components.

In this case the destruction of the reticular substance was extensive; therefore, the assumption seems to be justified that the fasciculus longitudinalis posterior had conducted the excitatory impulses which set up the nystagmus in the muscles innervated by the oculomotor nerve; however, the evidence is not conclusive because a certain number of reticular paths were intact. The question must be left open

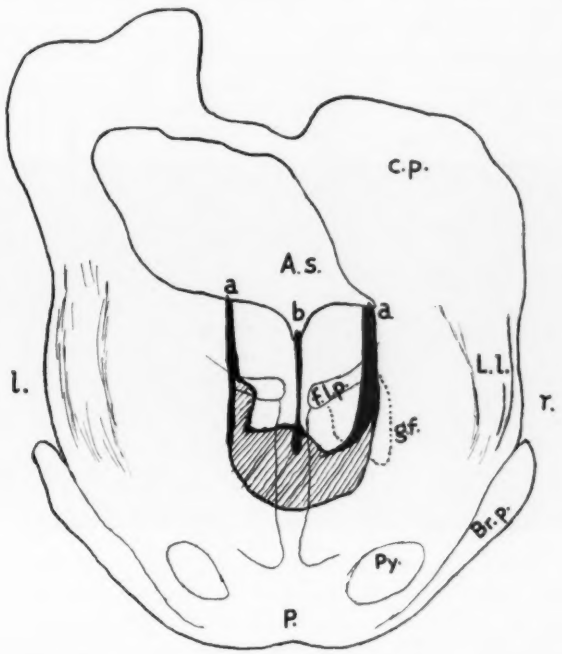

Fig. 17 (rabbit 5).—Transverse section through the posterior colliculus and pons: *b*, longitudinal lesion in the middle line; *a*, two lateral lesions made with a needle with the point bent in a right angle, which was rotated after the point had reached the desired depth. The whole lesion was enclosed in the midbrain.

until more evidence about the rôle of the fasciculus longitudinalis posterior impulses is available. On the basis of anatomy, which shows very numerous endings of these fibers on the motoneurons, it seems evident that the fasciculus longitudinalis posterior and tractus vestibulomesencephalicus must have an important function and transmit vestibular impulses to the motor cells; but as to the rôle played by these impulses in the normal rabbit, nothing can be stated with certainty. At any rate, it must be borne in mind that according to law 1, the fasciculus longitudinalis posterior impulses arrive at the motoneurons through numerous paths (fig. 10, *II*). This makes it almost impossible to obtain a preparation in which only fasciculus longitudinalis posterior impulses are being transmitted to the motor cells.

In former experiments [26] it was shown that the nystagmus reaction may be converted into a pseudopostural (i. e., monophasic) reflex by means of either a cross-section in the pons, as in experiment 4, or a longitudinal section in the middle line of the medulla and pons. After one of these operations the external rectus muscle, in spite of its connections with the primary vestibularis nuclei being intact, instead of the rhythmic nystagmus showed only steady relaxations or contractions of the same duration as the nystagmus itself would have had. This seems to prove that the primary vestibularis nuclei set up only a continuous stream of impulses, which in some relay are changed into a rhythmic series of excitatory and inhibitory impulses producing the rhythmic sequence of contractions and relaxations characteristic of the nystagmus. This view is also consistent with the fact that from the whole reaction to rotation (eye, head, body and limb reflexes) the nystagmus is the only reflex of rhythmic character. Since in the primary vestibularis nuclei no system of cells exists in connection only with the ocular muscles, evidently some internuncial relay located outside, i. e., in the reticular substance, has to take charge of the production of the rhythm. It has not been possible as yet to identify among

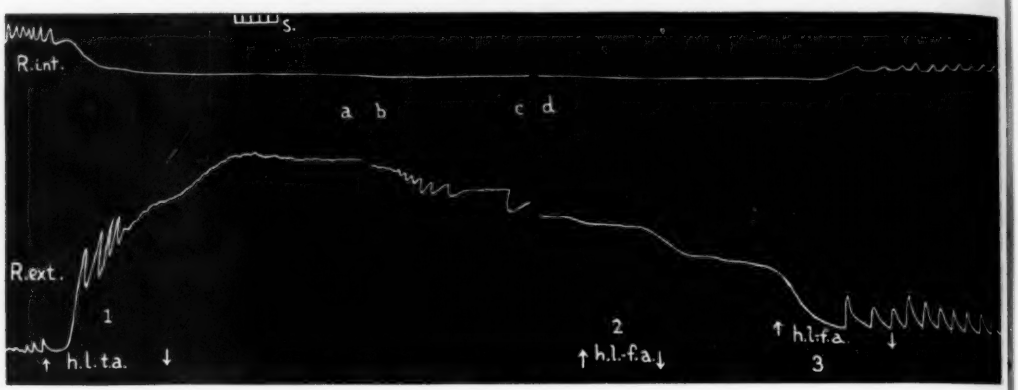

Fig. 18 (rabbit 5).—Some of the reactions of the horizontal muscles of the right eye to excitations of the left horizontal canal: *1*, current toward the ampulla. The after-discharge is so long that only a part of the tracings can be reproduced; between points *a* and *b* there is an interval of fifteen seconds and between points *c* and *d* another one of twenty seconds; *2*, the current from the ampulla which is able to cut down the after-discharge but not to reverse the direction of the nystagmus; *3*, the same current which is now able to set up the expected nystagmus.

the various anatomic structures destroyed by the aforementioned lesions those playing such an important rôle in the production of the nystagmus, but the former experiments and the following ones give some information that may be of value for the understanding of the problem.

EXPERIMENT 6.—*Feb. 22, 1932. Right eye, left and right horizontal canals.*

Operation.—A deep longitudinal lesion was made in the middle line of the medulla beginning at the level of the oral end of the motor nucleus of the trigeminus and ending a little caudal to the posterior end of the tuberculum acusticum. The lesion, exactly placed in the raphe, did not destroy any longitudinal pathway (fig. 19)

26. Lorente de Nó, R.: Die Grundlagen der Labyrinthphysiologie, Skandinav. Arch. f. Physiol. **49**:251, 1926; footnotes 6 and 16.

Results.—Only the external rectus reacted to labyrinthine stimuli; the reaction consisted of a pseudopostural reflex of abnormal direction (fig. 20). The current toward the ampulla in the right horizontal canal (*1*) produced first, as expected, a slow relaxation that, however, soon went over into contraction, increased further and, after withdrawal of the warm needle, remained as an after-reaction without decreasing during no less than fifteen seconds. At this moment a current from the ampulla (*2*) first caused an abnormal relaxation that soon changed into contraction and remained as an after-reaction; the current toward the ampulla (*3*) again produced first a normal relaxation, which soon changed into abnormal contraction and remained as an unusually long after-discharge.

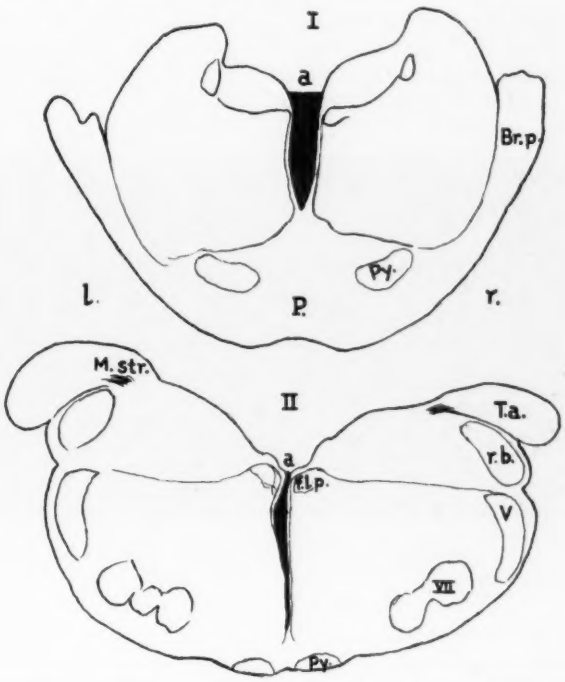

Fig. 19 (rabbit 6).—Transverse sections through the pons (*I*) and medulla (*II*) at the level of the caudal end of the facialis nucleus. *I* contains the anterior and *II* the posterior end of the lesion.

Comment.—This experiment proved that a longitudinal lesion cutting the pathways which cross the middle line between the level of the oral end of the motor nucleus of the trigeminus and the level of the caudal end of the tuberculum acusticum is enough to change the rhythmic nystagmus into a pseudopostural reflex. This result is in accordance with experiment 2 in which after a longitudinal section from the caudal end of the tuberculum acusticum backward the nystagmus still was present.

The primary vestibular nuclei themselves were not damaged in any way (except perhaps by the, in this case, irrelevant extirpation of the cerebellar vermis); if now these nuclei, as customarily accepted, had only efferent pathways and produced the rhythm of the nystagmus, the results of this experiment would be abso-

lutely impossible to explain. That the nystagmus became a pseudopostural reflex proves that for the production of the quick phase neurons are necessary that lie outside the floor of the fourth ventricle, and that the reaction of the rectus externus was abnormal proves that the section cut some centrifugal, i. e., recurrent, fibers ending in the primary vestibular nuclei and playing an important rôle in their function. This experiment adds evidence to the view already favored by the former experiments, that the reticularis relays and the recurrent collaterals constitute a fundamental element of the reflex arc.

EXPERIMENT 7.—*Jan. 23, 1932. Left eye, horizontal canals.*

Operation.—In the middle line of the pons, a longitudinal lesion (fig. 21, *I, a*) was made that began at the level of the posterior colliculus and reached the level of the posterior third of the abducens nucleus (fig. 21, *II, a*); up to the level of the facialis knee the lesion was very deep, but between the abducens nuclei it cut only the dorsal crossings. On the right side of the pons a transverse lesion was made (fig. *I, b*), cutting the homolateral pathways.

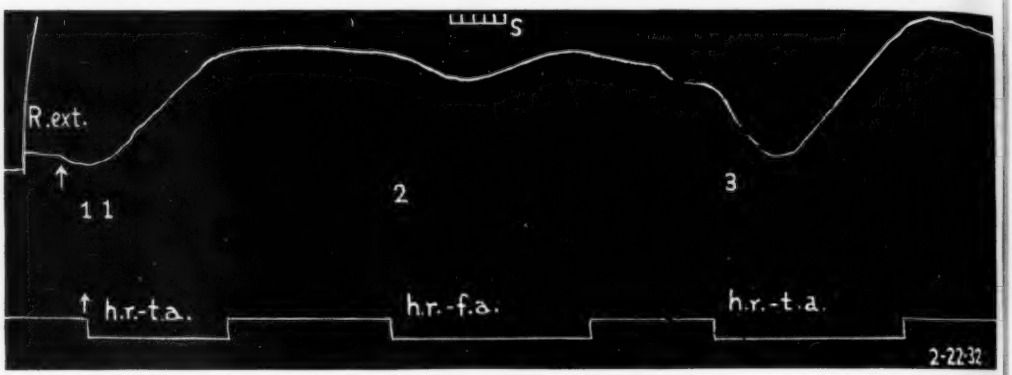

Fig. 20 (rabbit 6).—Reactions of external rectus muscle to excitations of the right horizontal canal.

Results.—Only three muscles (the superior, external and internal recti) reacted. The superior rectus showed small spontaneous nystagmus with quick contractions, which was reenforced when either a current toward the ampulla in the left horizontal canal or one from the ampulla in the right one was produced. The opposite currents did not modify it notably.

The horizontal muscles showed only the slow phase of the nystagmus in the expected direction; both labyrinths set up exactly the same reactions.

Comment.—The horizontal muscles had only the slow phase, but the superior rectus had a complete nystagmus. Similar results occurred often and seemed to prove that there exist certain reflex patterns in which each ocular muscle is represented as a unit.

Since the longitudinal section cut the crossings of the efferent secondary pathways from the oral third of the vestibularis nuclei, there is no doubt that the left and right labyrinth had quite different pathways for transmitting impulses to the left abducens nucleus; the right labyrinth had only the crossed ones of the inferior two-thirds, and the left labyrinth only the uncrossed ones. In spite of this, both labyrinths set up exactly the same reaction in the external rectus. Also the reflex

of the internal rectus set up by excitations of both labyrinths were alike, although on the right side the homolateral pathways were cut.

This fact, which repeats itself in some form or other in all experiments, has evidently no other interpretation than that the whole vestibular system, composed of all the primary and reticular nuclei on both sides owing to the existence of recurrent fibers and to the multiplicity of the pathways, constitutes a functional

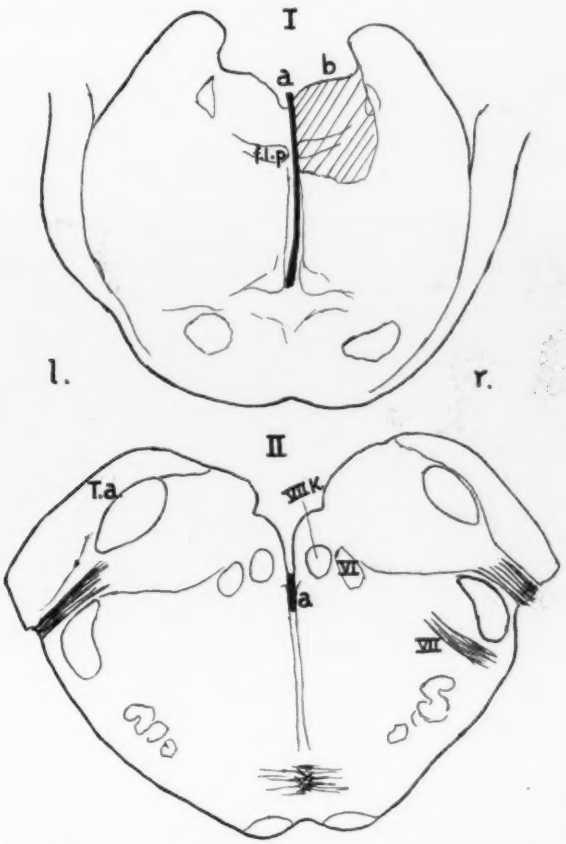

Fig. 21 (rabbit 7).—Transverse sections through the pons (*I*) and medulla (*II*): *a*, longitudinal lesion (anterior end in *I* and posterior end in *II*); section *II* goes through the caudal third of the abducens nucleus; *b*, projection of the transverse lesion in the pons.

unit, and when any part of it is destroyed the remaining structures, no matter from which labyrinth the impulses arrive, set up reflexes according to a pattern characteristic for the anatomic mechanism still able to function.

EXPERIMENT 8.—*Jan. 4, 1932. Left eye, left horizontal canal.*

Operation.—A deep longitudinal lesion was made in the middle line of the medulla, beginning at the level of the facialis knee and ending in the calamus scriptorius. The lesion was similar to that shown in figure 19, *II*.

Only the external and internal rectus muscles reacted to currents in the left horizontal canal with the slow phase of the nystagmus; the tracings were extremely similar to those in figure 22.

Comment.—This experiment is important not only because it again shows that if the section in the raphe extends through the whole length of the medulla oblongata the quick component of the nystagmus disappears in all muscles, but also because it proves that the homolateral pathways from the primary vestibular nuclei are enough for setting up a discharge from the muscles innervated by the oculomotor nerve.

In many experiments, after the homolateral pathways were cut, the reflexes were still present, disappearing after section of the crossings in the raphe. This experiment shows that if the crossed pathways from the primary vestibular nuclei are totally cut the internal rectus may still respond, which evidently proves that the homolateral paths, as well as the crossed ones, carry excitatory impulses.

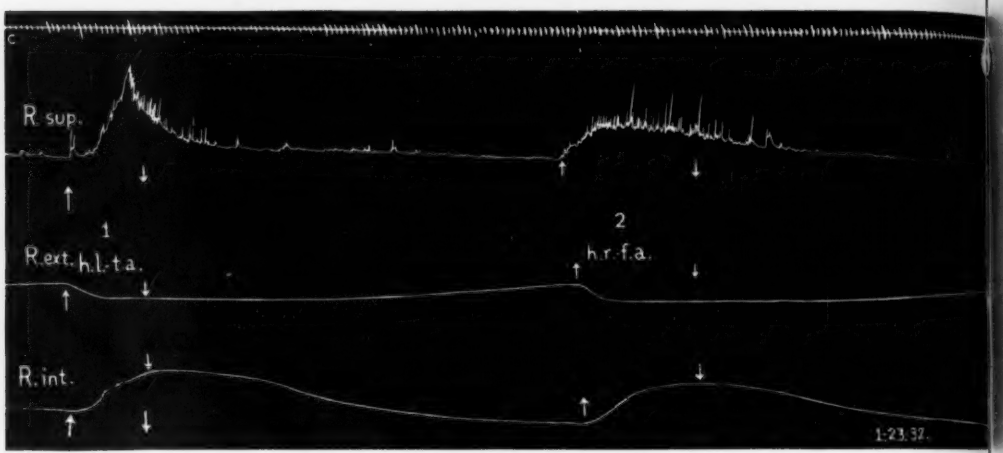

Fig. 22 (rabbit 7).—Some of the reflex reactions of the muscles of the left eye. The superior rectus shows a weak spontaneous nystagmus with quick contractions which notably increases when either a current toward the ampulla in the left horizontal canal (*1*) or a current from the ampulla in the right horizontal canal (*2*) is produced. The opposite currents do not modify this nystagmus notably. The horizontal muscles do not show any nystagmus. They respond to excitations of the canals only with the slow nystagmus component. Note that the currents toward the ampulla in the left horizontal canal and from the ampulla in the right one, in spite of the asymmetry of the lesions in the pons (fig. 21), are alike. The two other currents set up like reactions which are in the opposite direction to those reproduced in this figure.

EXPERIMENT 9.—*Jan. 5, 1932. Left eye, both labyrinths.*

First Operation.—A longitudinal section was made in the raphe of the medulla, beginning at the level of the posterior half of the abducens nucleus and reaching the calamus scriptorius; the lesion was very similar to that in figure 19, *II*.

A spontaneous nystagmus of a very peculiar kind arose; both horizontal muscles of the left eye showed quick contractions and slow relaxations and indeed very extensive ones (fig. 23, *Sp.*). In the right eye a nystagmus back and down-

ward was observed, which undoubtedly was being brought forth by similar contractions of the muscles.

The horizontal canals were able to modify only the frequency and amplitude of the spontaneous nystagmus but not to reverse it (fig. 23); the muscles also showed changes in tonus, but there was no rule determining the change produced by each current.

Second Operation.—A transverse lesion was made in the pons similar to that in figure 24, *I, a, b,* but much deeper.

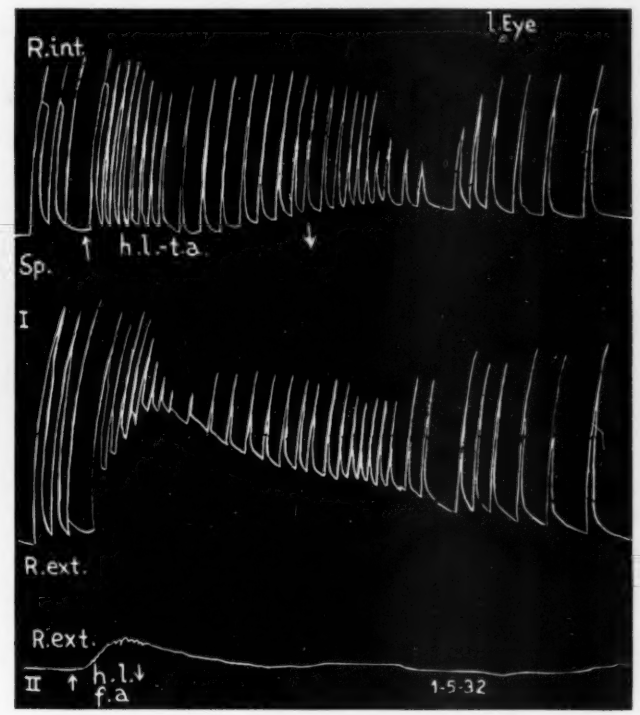

Fig. 23 (rabbit 9).—The spontaneous nystagmus and reflex reactions of the horizontal muscles of the left eye: *I,* after the first operation. *Sp.* represents spontaneous nystagmus composed of simultaneous quick contractions and simultaneous slow relaxations of both horizontal muscles; *h.l.-t.a.,* the current toward the ampulla in the left horizontal canal. The frequency of the spontaneous nystagmus increases and *R. int.* shows an increase in tonus, but no reversal of the spontaneous nystagmus is obtained. *II,* after the third operation. The excitations of the labyrinth set up in the external rectus nystagmus of normal direction. The figure reproduces the tracing of the nystagmus elicited by the current from the ampulla in the left horizontal canal.

The spontaneous nystagmus ceased.

Third Operation.—The vestibular nuclei on the right side of the medulla were destroyed by means of two longitudinal sections similar to those in figure 11.

The excitation of the left horizontal canal set up in the external rectus a nystagmus of small amplitude but of normal direction (fig. 23, *II*).

EXPERIMENT 10.—*Jan. 13, 1932. Left eye, both horizontal canals.*

First Operation.—Extirpation of the cerebellar vermis was done; a longitudinal lesion was made in the middle line of the medulla, as in experiment 9, but begin-

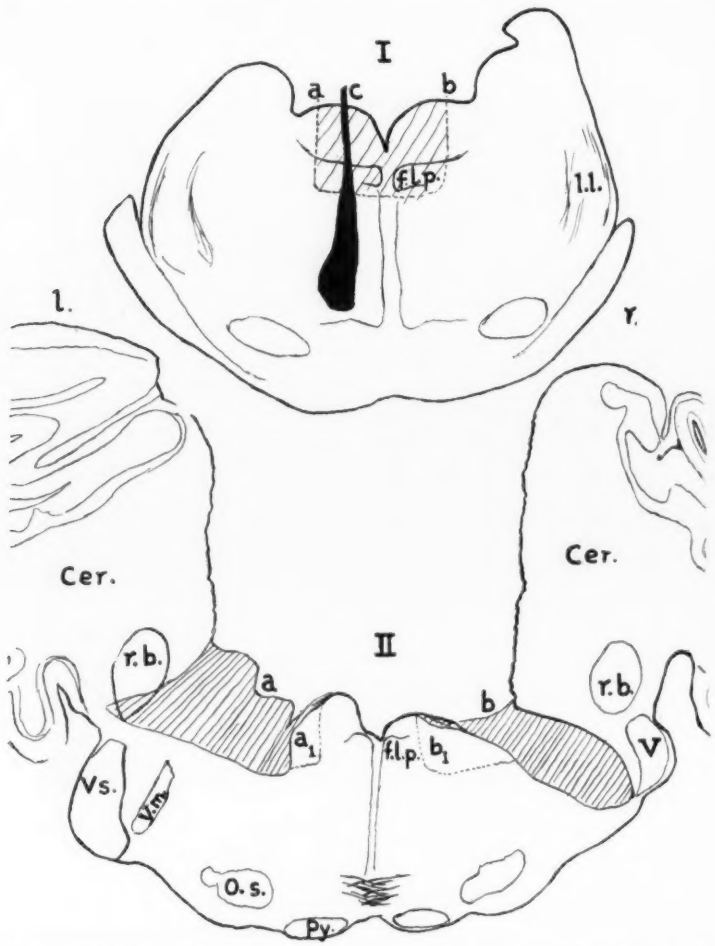

Fig. 24 (rabbit 10).—*I*, transverse section through the pons showing the lesions made in the second operation; *a-b* indicates the transverse lesion; *c*, the longitudinal lesion on the left side, which begins at the level of the motor nucleus of the trigeminus and reaches the level of the posterior colliculus. *II*, transverse section through the medulla showing the lateral lesions made in the first operation; *a* and *b*, parts of the lesions actually contained in the section; aa_1 and bb_1, the projection of the whole lesion.

ning a trifle caudal to the posterior end of the abducens nucleus, and two transverse lesions were made in the floor of the fourth ventricle, immediately oral and

lateral to the abducens nucleus (fig. 24, *II, a, b*), destroying the homolateral pathways on both sides.

Results.—The four rectus muscles showed a spontaneous nystagmus of the same kind as in experiment 9; in all the muscles quick contractions and slow relaxations were observed. The excitations of the labyrinth were able to modify only the frequency of the nystagmus, both labyrinths yielding similar results; only the current from the ampulla in the right horizontal canal was able to set up in the internal rectus a nystagmus of very small amplitude, but of normal direction.

Second Operation.—A transverse lesion was made in the oral extremity of the fourth ventricle, cutting the fasciculus longitudinalis posterior on both sides (fig. 24, *I, a, b*), and a longitudinal lesion was made on the left side of the pons (fig. 24, *I, c*).

The spontaneous nystagmus changed its nature; the vertical muscles remained quiet and the horizontal ones showed reciprocal nystagmus, the external rectus having quick contractions and the internal rectus quick relaxations. This nystagmus, however, was not continuous. Immediately after the operation it was strong, but it soon diminished in intensity and disappeared, to reappear again only as an abnormal reaction to the current toward the ampulla in the right horizontal canal; three hours after the operation it appeared only in the external rectus.

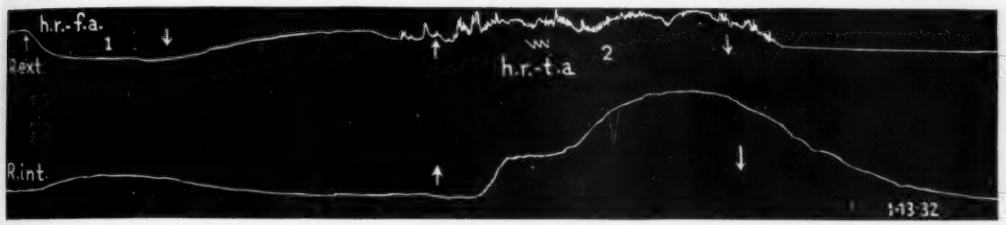

Fig. 25 (rabbit 10).—Reactions of the horizontal muscles of the left eye after the second operation. *1*, the current from the ampulla in the right horizontal canal sets up only the slow phase of the nystagmus in both muscles; the reaction has the expected direction; the currents in the left horizontal canal set up similar reactions, but the current toward the ampulla in the right horizontal canal (*2*) gives rise to an abnormal reaction; the external rectus shows a slight contraction and a very fast nystagmus with quick contractions, i. e., nystagmus of direction opposite to the one expected; the internal rectus shows a strong contraction, which also is totally abnormal.

The currents in the left horizontal canal and the current from the ampulla in the right horizontal canal set up in both horizontal muscles the slow phase of the expected nystagmus, the muscles being innervated reciprocally (fig. 25, *1*), but the current toward the ampulla in the right horizontal canal set up a totally abnormal reaction (fig. 25, *2*); the external rectus showed a tonic contraction and nystagmus of high frequency and small amplitude with quick contractions; the internal rectus first remained in a state of rest but after three or four seconds showed a strong contraction accompanied by nystagmus of small amplitude with quick relaxations. The expected reaction was contraction of the external rectus, accompanied by nystagmus with quick relaxations, and relaxation of the internal rectus accompanied by nystagmus with quick contractions, both muscles starting their reactions at the same time.

Comment.—Experiments 9 and 10 complement each other and also other experiments published elsewhere,[22] in which either a nystagmus with co-contraction in both horizontal muscles appeared or a nystagmus of reversed direction added itself to a normal slow phase. The nystagmus with co-contraction in experiment 9 could have been explained by the assumption that the homolateral pathways from each labyrinth continuously activate the motor centers of half of the muscles of each eye, this influence being compensated by that of the contralateral pathways of the other labyrinth. However, this view contradicts the results of former experiments in which a similar nystagmus appeared after exten-

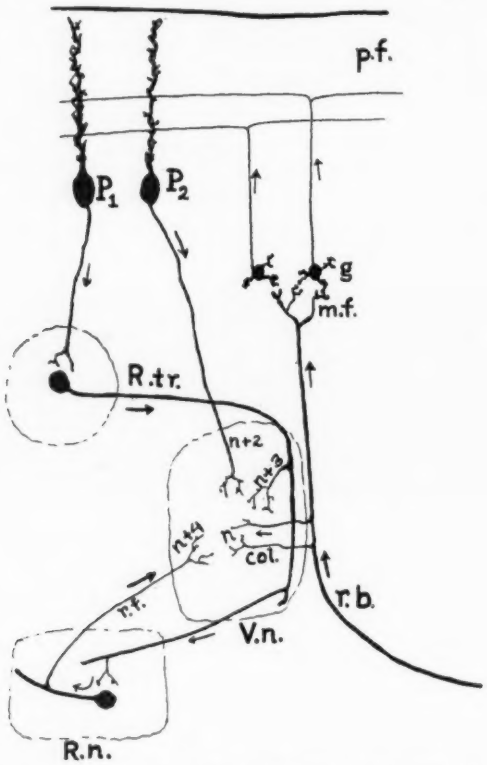

Fig. 26.—Diagram of the pathways that transmit the proprioceptive impulses carried by the restiform body to the vestibular nuclei: *r.b.*, restiform body with collaterals (*col.*) for the vestibular nuclei (*V.n.*); the restiform fiber has mossy endings (*m.f.*) in the cerebellar cortex; the granule cells (*g*) and the parallel fibers (*p.f.*) transmit the impulses to the Purkinje cells (P_1 and P_2). P_2 sends its axon to the vestibular nuclei; P_1, to the nucleus tecti which is the origin of Russell's tract (*R. tr.*). This tract gives off collaterals for the vestibular nuclei and ends in the reticular substance. The axons of the reticular cells give recurrent collaterals *r.f.* to the vestibular nuclei. In this diagram only the paths converging at the vestibular nuclei have been indicated. The vestibular nuclei have been drawn as a unit, whereas actually each kind of afferant fibers end in a different region of the nuclei. If one calls the impulses carried by the restiform body of n order, the other efferent fibers are of $n + 2$, $n + 3$ and $n + 4$ order (fig. 3, *1*).

sive destruction of the vestibular nuclei on both sides,[27] and also the results of experiment 10 in which although the homolateral pathways were destroyed the co-contraction nystagmus was present.

The co-contraction nystagmus must be interpreted as the result of an abnormal function of internuncial relays. Lesions outside the primary vestibular nuclei remove or fundamentally change the spontaneous nystagmus and in addition allow the primary vestibular nuclei to set up reactions of the normal direction again. Experiments 9 and 10 prove that the internuncial relays responsible for the co-contraction nystagmus lie in part in the midbrain, and in addition that they must work through the descending recurrent fibers. This agrees with the results of all other experiments. The appearance of reversed reaction to one current in spite of normal reactions to other currents is a phenomenon repeatedly observed; in this case (experiment 10), as well as in the former ones, the only explanation is to accept an abnormal activity in the partly damaged internuncial relays which, by means of the recurrent fibers, modify the activity of the primary nuclei. As a matter of fact, the abnormal reactions can be easily removed by further destruction of the reticular substance. Comparison of the lesions in experiments 7, 8, 9 and 10 shows that pathways of great importance for the establishment of the nystagmus must cross the middle line at the level of the anterior third of the abducens nucleus.

The mechanism determining the reciprocal innervation of the antagonistic muscles may not be considered as an anatomically fixed one; the reciprocal innervation depends on the functional state of the centers, which already seems to be evident if attention is paid to the fact that two ocular muscles can be innervated as antagonists or as agonists, depending on the kind of reflex action in which they are engaged.[28] The reciprocal innervation is therefore established, probably because the impulses reaching the motoneurons are coordinated in such a way that excitatory impulses do not arrive at the same time at antagonistic motoneurons. Other experiments seem to demonstrate that the relaxation of the antagonistic muscle is not due to an active inhibition of the motoneurons, but simply to the lack of excitatory impulses; i. e., that the motoneurons of the relaxed muscle are not in a state of inhibition, but in a state of rest.[29]

COMMENT

1. *After the long pathways (fasciculus longitudinalis posterior and tractus vestibulomesencephalicus) connecting the primary vestibular with the motor nuclei are cut, all the labyrinthine reflexes of the ocular muscles can still be elicited.* This fact has no little importance, because

27. Lorente de Nó, R.: The Regulation of Eye Positions and Movements Induced by the Labyrinth, Laryngoscope **42**:233, 1932.

28. Sherrington: Reflex Inhibition as a Factor in the Coordination of Movements and Postures, Quart. J. Exper. Physiol. **6**:251, 1913 (reciprocal innervation in spinal reflexes). A thorough analysis of the problem has been carried out recently by Ranson, S. W., and Hinsey, J. C.: Contralateral Flexor Reflex, Rebound Phenomena, Co-Contraction and Reciprocal Innervation in Spinal and Decorticate Cats (Arch. Neurol. & Psychiat. **26**:247 [Aug.] 1931); they also review the literature. Lorente de Nó.[27]

29. Lorente de Nó, R.: Interaction of Corneal Reflex and Vestibular Nystagmus, Am. J. Physiol. **103**:704, 1933.

the posterior longitudinal bundle has always been described as the pathway that closes the vestibulo-ocular arc.

According to current ideas on the interpretation of the results obtained with the so-called extirpation technic, the conclusion should be drawn that the fasciculus longitudinalis posterior and the tractus vestibulomesencephalicus do not constitute an important part of the vestibular mechanism. But this would evidently be a mistake. There can be no doubt that the fasciculus longitudinalis posterior conducts impulses to the motoneurons and upper reticular relays, and it is an important problem to determine what modifications appear in the labyrinth reflexes when these impulses are missing. The technic used in the present work, in which movements of the ocular muscles are recorded directly, is undoubtedly much more reliable than the customary one consisting in the examination of the movements of the eye as a whole, but it is still insufficient; experiments in which the tension developed by the muscles and the time relations of the reflexes are measured by means of isometric myographs are now being carried out.

None of the experiments as yet published furnish evidence directly justifying any conclusion as to the rôle played by the impulses that the fasciculus longitudinalis posterior and tractus vestibulomesencephalicus conduct to the motoneurons, but indirectly it is possible to reach the following interpretation, which has already been advanced in other papers.[22]

The fasciculus longitudinalis posterior impulses, if working alone, should not be able to set up a motor discharge but only a facilitation of the motoneurons. Two facts speak in favor of this view: (1) The fasciculus longitudinalis posterior being intact (and the motoneurons not in state of inhibition as evidenced by effective reflexes over other afferents), the motor reactions may be absent (experiment 1); (2) the arcs closed through the substantia reticularis enclose at least one neuron more than the fasciculus longitudinalis posterior arc (fig. 8). Fact 1 has only two possible explanations: since the motoneurons themselves are not in a state of inhibition, it follows that either the fasciculus longitudinalis posterior impulses are subliminal or they are not produced because the neurons of the primary vestibular nuclei themselves are in a state of inhibition. This second explanation is unlikely but not impossible. Fact 2 has the following significance: The fasciculus longitudinalis posterior arc includes only one internuncial neuron, while the reticular arcs have at least two; among the reticular arcs there are several, those closed through the nucleus interstitialis or the para-ocular nuclei having the first neuron in common with the fasciculus longitudinalis posterior; since in each neuron a synaptic delay (Sherrington) takes place, the fasciculus longitudinalis posterior impulses will probably arrive at the motoneurons some time before the

reticular impulses. If now the fasciculus longitudinalis posterior impulses had set up a motor discharge, what could be the rôle of the reticular impulses which would find the motoneurons in a refractory state? Many assumptions could be made, but the simplest one is to suppose that the fasciculus longitudinalis posterior impulses are subliminal and therefore do not set up a motor discharge until the reticular impulses arrive and reenforce by summation the excitation already produced.

This is a problem concerning the general physiology of nerve cells. On each cell in the nervous system numerous synapses, sometimes several thousand,[14] are found. The synapses are always of different kinds, occasionally of ten or more. May one class of synapses set up a discharge of the cell, or is the coincidence of the excitation of several kinds of synapses necessary; if so, how many impulses must arrive at the cell, and in how much time and in what sequence?

On the other hand, still another question appears. The descending fibers of the nucleus interstitialis establish connections with the internuncial relays and the primary vestibular nuclei (fig. 8, *II*); the experiments (for instance, experiments 5, 6 and 10) show that the impulses conducted by these fibers modify the function of the vestibular nuclei, i. e., modify the fasciculus longitudinalis posterior impulses themselves. But while this reciprocal influence is being produced the motoneurons are discharging. The problems thus stated seem to have no other explanation than that *the whole vestibular system is a physiologic unit that finds itself in constant activity, and according to its functional state the afferent impulses set up reflexes of determined pattern, because they find open only a limited number of the extremely numerous anatomic paths.*

2. *The reflex after-discharge,* i. e., the reflex reaction outlasting the peripheral excitation, *is a "central" phenomenon.* In earlier times of physiologic research it could be accepted that the individual neurons store up the excitation and expend it gradually. But Forbes [30] forcibly stated that, since the neurons discharge according to the all-or-nothing law, no storage of excitation by a single neuron is possible and, therefore, as long as the reflex reaction is taking place the motoneurons must be under a constant bombardment by excitatory impulses. Forbes' assumption has become one of the fundamental concepts in the modern physiology of the nervous system.

The difficulty arises when the source of the constant stream of excitatory impulses is sought. Forbes tentatively advanced an explanation known under the name of "delay paths theory."

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30. Forbes, A.: The Interpretation of Spinal Reflexes in Terms of Present Knowledge of Nerve Conduction, Physiol. Rev. **2**:361, 1922.

According to Forbes, the afferent impulses should have at their disposal many different paths, all converging at the motoneurons. Some paths should be simple and some others ("delay paths" of Forbes), which include internuncial neurons, complicated. The afferent impulses travel through the simple paths in a short time, which is the latent period of the reflex; but to travel through the most complicated delay paths takes a longer time, which corresponds to the duration of the reflex discharge. Between the first and the last ones a continuous series of impulses arrive at the motoneurons through progressively more complicated pathways.

This suggestive theory has to meet several serious difficulties. Fulton [2] has already mentioned some of them, the most important being that delay paths explaining long after-discharges do not seem to exist. Forbes, Davis and Lambert [31] then put forward the complementary assumption that the impulses might go through the same delay paths several times.

The "delay path" theory is based on two fundamental laws: the "avalanche conduction" (Cajal), according to which a single impulse arriving through an afferent fiber may be transmitted to numerous cells and these in their turn again excite a much higher number of cells, etc., and the "principle of the final common path" (Sherrington), according to which all the pathways converge at last at the motoneurons.

But the "avalanche conduction" is an anatomic theory for which physiologic evidence has not yet been ascertained. If the afferent impulses should find all the anatomically possible ways open, the excitation would diffuse through the whole nervous system; since the excitation remains confined to certain pathways it is evident that not all of them are open, perhaps because the afferent impulses fail to excite above threshold some of the neurons they reach or because certain neurons are in a state of inhibition. On the other hand, the principle of the final common path does not exclude the fact that the converging pathways at the same time excite the particular motoneuron as well as many others. Therefore, if the impulses should travel through a progressively increasing number of transmission systems, the reflex reaction would progressively engage new motoneurons. These two physiologic facts, (1) that the reflex reaction does not change qualitatively from beginning to end (provided, of course, that the excitatory impulses are not mixed ones) and (2) that it remains confined to the same muscles, together with the anatomic fact that the delay paths establish connections with a progressively increasing number of internuncial neurons and with many other reflex arcs and trans-

31. Forbes, Davis and Lambert: The Conflict Between Excitatory and Inhibitory Effects in a Spinal Center, Am. J. Physiol. **95**:142, 1930.

mission systems, offer to the original form of the theory of delay paths an insurmountable obstacle.

To avoid this obstacle, the theory of closed "self-reexciting" chains of neurons has been put forward,[32] *a theory that also fulfils the fundamental condition of explaining the after-discharge by means of a constant bombardment of the motoneurons by excitatory impulses as postulated by Forbes.* In it the concept of neuron threshold as developed by Sherrington [33] plays a preeminent rôle. According to Sherrington, when excitatory impulses arrive at the neurons a special state, the central excitatory state capable of gradation and summation, is created. When the central excitatory state, which may be compared with the local excitatory state of the peripheral nerves (K. Lucas), reaches a certain value—the threshold value—the neuron discharges.

The theory of closed self-reexciting chains is based on laws 1 and 2 stated at the beginning of this paper. In law 2 (fig. 3, *II* and *III*) *A* and *B* may be thought of as being single neurons or else as being one or several superimposed chains (fig. 3, *I*, *N*, *n*, $N + 1$). When *A* discharges the impulses go toward the effector system through the path *a* or *b*, and at the same time come back through path *r.f.* and reexcite *A*, etc. The cyclic process continues until either *A* or *B* fails to discharge. According to this view the fundamental factors in the nervous system are neither the single neurons nor the elementary reflex arcs, but the chains of neurons of the type discussed, which are capable of rhythmic activity.

The smallest part of the vestibular system still able to set up motor discharges has been found to be limited by the following four sections: [16] (1) a cross-section through one-half of the medulla immediately oral to the abducens nucleus; (2) a cross-section through the same half of the medulla through the posterior third of the tuberculum acusticum; (3) a longitudinal section of the medulla in the raphe; (4) a horizontal section passing immediately above the tuberculum acusticum through the ascending vestibular root and disconnecting the cerebellum. This

32. Lorente de Nó, R.: Researches on Labyrinth Reflexes, Tr. Am. Otol. Soc. **22**:287, 1932. (After the publication of this paper I observed a paper by S. W. Ranson and J. C. Hinsey [Am. J. Physiol. **94**:472, 1930] in which a similar theory had been put forward. Ranson and Hinsey's theory of "reverberating circuits" accepts the existence of closed rings constituted by premotor neurons linked together by collaterals of their axons; premotor neuron 1 excites a motoneuron and neuron 2; neuron 2 excites another motoneuron and neuron 3 and so on; the last neuron excites a motoneuron and again neuron 1). R. Bárány (Theorie des kortikalen Mechanismus der Assoziation [des bedingten Reflexes] und des Sukzessivvergleichs, Deutsche Ztschr. f. Nervenh. **124**:216, 1932) also accepts the existence of closed circuits of neurons in the cerebral cortex.

33. Sherrington, C. S.: Remarks on Some Aspects of Reflex Inhibition, Proc. Roy Soc. London, s.B. **97**:519, 1925; Some Functional Problems Attaching to Convergence, ibid. **105**:332, 1929; Quantitative Management of Contraction in "Lowest Level" Co-Ordination, Brain **54**:1, 1931.

small portion of medulla contains all the elements necessary for the production of the slow phase of the nystagmus in the external rectus muscle. The after-discharge is generally somewhat shorter than normal, but there are cases in which (after rotations) it is almost as long as in the normal rabbit.

The nerve cells and pathways still in function are indicated in figure 8, *I; A* represents the neurons with a long axis-cylinder of Deiters' nucleus and of the nucleus of the descending root; *B*, the neurons with a short axis-cylinder in the same nuclei; *C*, the neurons in the angular and ventromedial nuclei; *R*, the neurons in the reticular substance, and *M*, the motoneurons (*C* and *R* actually are chains of neurons).

One may think that the impulses are conducted in the following way: The discharge of *A* facilitates, i. e., excites subliminally, cells *B*, *C* and *M* and sets up a discharge of *R;* the discharge of *R* sets up a discharge of *C* and of the already facilitated cell *M;* the discharge of *C* sets up a discharge of the already facilitated cell *B* and facilitates *R* and *A;* finally, the discharge of *B* sets up a discharge of cell *A*, and the cyclic process continues until one of the cells, *A*, *B*, *C* or *R*, fails to reach a threshold excitatory state.

The physiologic interpretation of the anatomic structures is in accord with the results of all the experiments reported before. The characteristics of the reflexes have been found to be dependent on the reticular relays (*R*) as well as on the primary vestibular nuclei (*A, B, C*). The vestibular nuclei (*A, B, C*) and their unbroken pathways (*f.l.p.* and *n.v.m., a*, fig. 8) being intact, the muscle reactions may be absent if the reticular substance (*R*) is damaged. The duration of the after-discharge depends on the state of the reticular relays (*R*). If the whole vestibular system is considered, it is found that the chains of neurons cut or disconnected by the fourfold operation described constitute a system of collateral chains superimposed on the fundamental one represented in figure 8 or still better on the chain *A, B*.[34] This elementary chain receives the afferent vestibular impulses and sets the whole machinery into activity. It is a fact that the afferent impulses set up a reflex reaction and no matter how long they last, seconds, minutes or hours,[35] the reflex reaction remains unchanged, and when the afferent impulses are interrupted the same reflex reaction still continues (after-discharge). Therefore, it must be accepted that the same cells have been engaged during the whole time; i. e., that there may be many anatomic pathways through which the vestibular impulses might travel toward the motoneurons, but only a certain number of them are open, and once a chain has been activated the impulses remain within it. That the vestibular impulses really have at their disposal different anatomic paths is demonstrated by the fact that no labyrinthine excitation necessarily sets up a determined reaction.[36] This has been shown with the normal

34. That the cerebellum has to be considered as a system of collateral chains superimposed on the vestibular or other reflex arcs and therefore may not be considered as the "center" of any reflex was shown in another paper (Lorente de Nó, R.: Etudes sur le cerveau postérieur: III, Trab. d. Lab. de Invest. biol. Univ. de Madrid **22**:51, 1924).

35. By means of caloric stimulation it is possible to maintain the nystagmus, without any change, for at least fourteen hours.

36. M. H. Fischer has arrived at similar conclusions (Fischer, M. H.: Ueber die Beeinflussung einiger calorischen Vestibularisreaktionen durch zentral-nervöse Faktoren, Ztschr. f. Hals-, Nasen- u. Ohrenh. **29**:217, 1931. Fischer, M. H., and Oldberg, E.: Abhängigkeit der calorischen Vestibularisreflexe vom Zustande der nervösen Zentren, ibid. **30**:499, 1932). Lorente de Nó.[16, 12b]

rabbit, as well as after operations on the medulla, pons or midbrain. The rôle of the collateral chains must be partly that of determining which of the elementary neuron chains *A-B* are open to the afferent impulses.

In a similar way the state of the reticular relays modifies the function of the vestibular nuclei according to the impulses concurrent with the vestibular ones; this would explain the reflex reversal conditioned by extralabyrinthine impulses; and it is now important to note that any other kind of impulses, for instance, from the receptors of the body musculature, will arrive at the vestibular nuclei through many different paths, i. e., through chains of neurons of the same fundamental structure as those in figures 3 and 8. If one consider only Flechsig's tract, the proprioceptive impulses ascend to the cerebellum through the restiform body (*r.b.*) (fig. 26) and descend again to the vestibular nuclei through the cerebellar fibers, either from the cortex or from the nucleus tecti (*R. tr.*); but before they reach the cerebellum they have already facilitated Deiters' nucleus by means of the collaterals of the restiform body (*col.*). In the same way the impulses conducted by the tractus uncinatus (*R. tr.*) excite the vestibular nuclei first directly and then through the recurrent collaterals from the reticular substance (*r.s.*).

But it must not be forgotten that this is a tentative explanation which, to be sure, new experiments will prove to be at least incomplete. At the present moment the value of the theory consists in offering an easy method of remembering all the physiologic facts that have to be accounted for. The greatest difficulty seems to be to explain how the excitation remains confined to certain chains of neurons, i. e., why many anatomic ways remain closed to the afferent impulses. Undoubtedly inhibitions take place, but as yet no satisfactory explanation of the central inhibition exists. Provisionally, it can be accepted that if a chain of neurons comes into activity, activation of antagonistic chains is prevented by inhibition of some cells. How in certain cases, even without inhibition, the cells with a short axis-cylinder might account for it has been discussed in another paper.[14]

Only one point more will be mentioned: According to this theory the duration of the after-discharge will depend on the time relations within the closed chains, i. e., on the (Sherrington's) threshold of the single neurons of the chain; if the threshold of the cells is raised, the after-discharge is likely to disappear soon, but if the threshold is lowered the after-discharge will last longer. Experiment 5 is a good example. After destruction of a good deal of the reticular pathways the after-discharge was enormously increased. Similar examples have been reported elsewhere.[22]

3. *After certain lesions in the medulla, the nystagmus is converted into a pseudopostural reflex* having the direction of the slow phase and apparently the same duration as the nystagmus would have had. This may help to understand the nature of the nystagmus.

Generally it is accepted that the two components of the nystagmus are of fundamentally different nature. The quick phase, also called the compensatory one, should have a central origin. This idea, which was outlined by Breuer, was explicitly expressed by Ewald and after-

ward, in some or other form, was accepted by all investigators.[37] Only recently [6, 22] the view has been advanced that the nystagmus is nothing else than an alternating reflex, perfectly comparable with the known rhythmic reflexes, for instance, the scratch reflex. The labyrinthine excitation is a complex one and determines all the characteristics of the ocular nystagmus, those of the slow phase (speed) as well as those of the quick one (amplitude), in the way, for instance, the excitation of the skin determines the characteristic of the scratch reflex. Spiegel [23] also favors a similar view when he establishes the comparison between nystagmus and reflex stepping as studied by Graham Brown in the deafferented spinal preparation.

The mechanism of production of rhythmic reflexes has not been explained yet. In old times numerous theories were enunciated (reviewed recently by Graham Brown [4]), but as the all-or-nothing character of the neuron discharge could not be considered they have today little value. In modern times emphasis has been laid on two facts: [38] (*a*) that the reflex movement is operated by two sets of antagonistic muscles which, according to the principle of reciprocal innervation, contract and relax in alternating sequence, and (*b*) that if two antagonistic nerves are tetanically excited the muscles involved in the reflex reaction, instead of remaining in a state of rest, may show alternating series of contractions and relaxations similar to those appearing with the rhythmic reflexes (discussion in Forbes [30] and Ranson and Hinsey [28]).

Graham Brown put forward the following tentative explanation: Two antagonistic cells *A* and *B* find themselves under a continuous excitatory strain. *A*, by means of collaterals of its axon, can inhibit *B* and, reciprocally, *B* can inhibit *A*. When *A* discharges, *B* is inhibited, but after a certain time *A* becomes fatigued; the inhibition of *B* is released, but this cell begins to discharge, producing the inhibition of *A;* therefore, the reflex reaction is reversed. When *B* becomes fatigued, *A* again comes into play and a new reversal of the reflex reaction takes place, and so on until the excitatory strain disappears.

Graham Brown thought that the rhythm could be produced in the motor nuclei themselves, the motor collaterals being the inhibitory fibers. Forbes and Ranson found it more advisable to accept the existence of internuncial neurons in the rhythmic mechanism, and Spiegel in his theory of nystagmus thinks also that internuncial neurons should play the fundamental rôle.

Spiegel's [23] theory is the following: He denies that the rhythm of the nystagmus can be produced in the motor nuclei. He also denies that the substantia reticularis can play an important rôle, and accepts the theory that the rhythm is

37. It is interesting to note that Sherrington as long ago as 1912 (Quart. J. Exper. Physiol. **6**:284, 1912) took for granted that the nystagmus was a reflex of the same kind as the scratch reflex, etc. However, in the literature on the labyrinth, nystagmus has always been described as a reflex *sui generis.*

38. Creed, R. S.; Brown, D. D.; Eccles, I. C.; Lidell, E. G. T., and Sherrington, C. S.: Reflex Activity of the Spinal Cord, London, Oxford University Press, 1932, p. 142.

produced in the primary vestibular nuclei. Here two kinds of neurons should be present, *R* (right) cells which set up the contraction of the eye muscles producing an eye movement to the right, and *L* (left) cells which set up the contraction of the muscles that move the eye toward the left. The *R* cells will set up, among others, a contraction of the right external rectus; the *L* cells a contraction of the right internal rectus. When the *L* cells are activated, collaterals of their axons set up, by "Wedensky's interference," an inhibition of the *R* impulses. When the *L* cells are now submitted to a continuous series of impulses they will first set up a contraction of the internal rectus and through inhibition of the *R* impulses a relaxation of the external rectus. But the *L* cells will soon become fatigued and cease to produce impulses; the internal rectus will, therefore, relax, and the external rectus, since the *R* impulses are no longer inhibited, will contract. When the *L* cells recover the process reverses, etc. According to this theory the rhythm of the reflex is, therefore, produced by a rhythmic sequence of states of fatigue and recovery of the cell *L* under a constant excitatory strain, which is unlikely.

The theory of fatigue may perhaps offer a relatively satisfactory explanation of the rhythmic reactions produced by concurrent stimulations of antagonistic nerves, but it is scarcely able to explain the physiologic reflexes in which only a peripheral excitation is present; besides, these reflexes to a certain extent are independent of reciprocal innervation. Nystagmus evidently is accompanied by reciprocal innervation, but nystagmus may subsist with co-contraction (experiments 9 and 10) and also when all the motor nuclei but one (the sixth nucleus) have been extirpated. Reciprocal innervation is, therefore, something additional to the rhythmic mechanism, but not indispensable. On the other hand, lesions outside the vestibular nuclei convert the nystagmus into a pseudopostural reflex, and this fundamental fact remains totally unexplained by the theories mentioned.

This fact fits only in a theory put forward by Bárány in 1907.[39] Bárány observed a patient in whom as a consequence of a lesion in the pons close to the abducens nucleus the quick component of the nystagmus as well as the voluntary movements of the eye were absent. Only the slow phase of the nystagmus could be elicited. Bárány, therefore, accepted the existence in the pons of a center of higher category taking charge of both functions, the transmission of the volitional impulses and the production of the quick phase. The vestibular nuclei should produce the slow phase, and the center in question the quick one. As at the time the connections of the vestibular nuclei with the reticular substance were still unknown, Bárány was forced to assume an automatic activity under cortical control of the center of the quick component. This evidently was the weak link in the theory and perhaps the reason why it failed to find wide acceptance.

39. Bárány, R.: Physiologie und Pathologie des Bogengang-Apparates beim Menschen, Vienna, Franz Deuticke, 1907.

The fact that after determined lesions the nystagmus may be converted into a pseudopostural reflex, having the direction of the slow phase and the same duration as the nystagmus itself, seems to put two conclusions beyond reasonable doubt:[40] (*a*) that the vestibular nuclei up to a certain point set up a continuous series of excitatory impulses and (*b*) that additional neurons are necessary for the production of the rhythm. Experiments 4 and 6 and similar ones demonstrate that these neurons at least partly are located outside the primary vestibular nuclei. One can then accept two neuron systems *V. N.* (vestibular nuclei) and *Q* (fig. 27). The group *V. N.* sets up a continuous series of impulses that produce the discharge of the motor cells *M* and at the same time excite the neuron system *Q*. After a certain time *Q* discharges and inhibits the effect of the *V. N.* impulses; therefore, muscle *M* relaxes. After the discharge, *Q* enters into a refractory state, the inhibition ceases, and, therefore, the impulses *V. N.* again set up a contraction *M* which increases until *Q* discharges. The cyclic process continues as long as *V. N.* impulses are produced. For the antagonistic muscle, *V. N.* will set up relaxations and *Q* contractions.

If now it is accepted that the *V. N.* impulses are similar to those setting up "recruitment" reflexes (Sherrington) and the *Q* ones similar to the "d'emblée" impulses, the difference in the speed of both alternating phases is easily explained.

Neuron system *Q* must set up impulses with latency time equal to the duration of the slow phase of the nystagmus; this latency time is not constant, but dependent on the intensity of the series of *V. N.* impulses in much the same way as the latent period of the spinal reflex arcs is dependent on the intensity of the afferent impulses (discussion in Eccles and Sherrington[41]). If the slow phase has a great speed, the quick phase appears earlier; the same thing happens if impulses of another kind (neck reflexes) concurrently excite neuron system *Q*.

Examples of neuron systems functioning in a similar way are known; for instance, Adrian and Buytendik described in the ganglia of insects a slow negative wave preceding the discharge of the rhythmic series of motor impulses; similar slow potential changes have been described in mammals by Berger, M. H. Fischer, Bishop and Bartley (cerebral cortex) and Gasser and Graham[7] (spinal cord).

The first question that now arises is whether neuron system *Q* acts on the motoneurons themselves or through some internuncial neuron.

40. On the other hand, the assumption of Graham Brown and Sherrington that in the rhythmic reflexes a constant excitatory background is rhythmically interrupted by inhibitory impulses receives a strong support.

41. Eccles and Sherrington: Studies on the Flexor Reflex: I. Latent Period, Proc. Roy. Soc., s. B., London **107**:511, 1931.

Experiments in which the corneal reflex was pitted against a preexistent vestibular nystagmus favor the second view (fig. 27, *II*).

As to the constitution of the system *Q*, little can be said. There is no doubt that it cannot be an anatomically well defined nucleus, because extensive lesions are needed in order to suppress the quick nystagmus component in all the muscles. Comparison of the lesions in rabbits 6 and 8 shows that system *Q* must be located in the neighborhood of the abducens nucleus, or better said, that in this region important elements are present which are necessary for the transmission of the impulses to the motor nuclei. In some experiments (not reported in this paper), after a longitudinal lesion placed exactly between both abducens nuclei the quick nystagmus component of the horizontal muscles disappeared while the four other muscles still were showing nystagmus, in a manner similar to what happened in rabbit 7. Transverse lesions suppress the quick nystagmus component only when they lie immediately oral to the abducens nucleus muscles (*f.p.*), but if combined with short longitudinal lesions they may be placed in the pons. But whether the nystagmus disappears because the afferent fibers of *A* have been cut, or because *Q* has lost its efferent fibers or else because the endogenous fibers of the *Q* systems (right and left) have been cut, is a problem that only further research may solve.

4. *The experiments reported in this paper exclude the possibility of "localizing" the reflexes in anatomic nuclei, i. e., make it impossible to accept that each labyrinthine reflex is represented in a determined anatomic structure different for each reflex and located in a special region of the medulla or midbrain* (Magnus [21]). The whole vestibular system has revealed itself as constituted by numerous chains of neurons, reciprocally connected in many ways and having their links in various anatomic nuclei. All the chains work in intimate collaboration and all are necessary for the production of the normal reflex reaction. It has been found, as a rule, that whenever a lesion is made in any part of the anatomic mechanism, the reflex reaction becomes abnormal and therefore loses its purposive character. As the function of the nervous system is to set up not only "reflexes" but "physiologic reflexes," it is evident that any part of the nervous system able to modify the reflex reaction belongs to the "reflex mechanism."

On the other hand, even in those anatomic structures that are necessary for setting up the "skeleton" of the reflex, no localization is possible. The reflexes can be elicited as long as the reflex arc remains closed, and the experiments show that many possibilities exist. For instance, if the crossing efferent pathways of the anterior third of the primary vestibular nuclei are cut, the horizontal canal makes itself effective on the abducens nucleus on the contralateral side by means of the

crossing pathways of the posterior two thirds of the vestibular nuclei; but if the crossing pathways of the posterior two-thirds are cut, the horizontal canal produces a similar reaction by means of the crossing pathways of the anterior third. If the homolateral pathways are cut, the labyrinth may let the internal rectus contract by means of the crossed ones, but if the crossed pathways are cut the labyrinth uses the uncrossed ones, etc. (experiment 8). The conclusion that must be drawn from all these facts is that in the intact rabbit impulses travel at the same time through the different paths, and it is the result of their interaction which determines the characteristics of the reflex reaction.

SUMMARY AND CONCLUSIONS

Studies on the anatomy of various regions of the nervous system, using Golgi's and Cox's methods, have shown that the customary concept of the reflex arc is insufficient because it does not take into account two fundamental elements, the cells with short axis-cylinders and the recurrent fibers (collaterals of the axons and centrifugal fibers). These two elements were thought of as existing only in special cases, but when sought by these methods they have been found everywhere. Since the impulses that travel down the axis-cylinders pass into all the collateral branches, it follows that the transmission systems are made up of complicated circuits which always are of one of the two types indicated in figure 3.

The plan of structure of the trigeminal reflex arc is indicated in figure 5. On the basis of only anatomic data it is impossible to assume which paths are used by the impulses that set up the motor reflexes. Since experimental work on the trigeminal system is extremely difficult, the vestibulo-ocular arc has been analyzed both anatomically and physiologically.

Anatomically, it has been found that in the vestibular nuclei numerous hitherto unknown types of cells with a short axis-cylinder are present; the extremely numerous fibers from the vestibular nuclei to the reticular substance have been identified and the recurrent collaterals from the reticular substance and secondary vestibular paths to the primary vestibular nuclei have been described.

In the vestibular system, laws 1 and 2 (fig. 3) are observed; several examples of circuits obeying these laws have been indicated in figures 8 and 26. The experiments demonstrate that the numerous pathways between the vestibular nuclei and the motoneurons are always being used at the same time and that, therefore, the reflex reactions are not determined in one nucleus, but rather are the consequence of the interaction of all the nuclei and the paths of the whole vestibular system. It must be considered not only that the peripheral labyrinth is a physiologic unit, but also that its centers constitute a functional unit.

The vestibular centers or at least a certain part of their neurons are always in automatic activity, which chiefly is conditioned by the constant (postural) labyrinthine stimuli; but as Leidler,[42] Spiegel [43] and I [26] have corroborated by experiment, this activity is partly automatic and remains after extirpation of the labyrinth. The constant activity of the centers determines to a certain extent which chains of neurons are open to the afferent impulses; this explains the reflex reversal and agrees with the concept of neural balance developed by Graham Brown.[44] The reflex reversal is produced in the primary nuclei as a consequence of the activity of reticular relays.

Among the neuron chains there are several types of closed circuits, called self-reexciting chains, which are capable of rhythmic activity and which, therefore, may set up a prolonged after-discharge. The concept of closed self-reexciting chains is an elaboration of Forbes' theory of delay-paths; [30] similar theories have been proposed by Ranson and Hinsey and by Bárány.[32] A diagram is given in figure 8 which is supposed to indicate the fundamental functional plan of the vestibular system. In this diagram it is assumed that the reticular substance is necessary for the maintenance of the after-discharge, but possibly the chains within the primary nuclei (cells *A, B, C,*) are sufficient for this effect. Further work on this problem is under way.

Nystagmus is nothing else than an alternating reflex similar to other known rhythmic reflexes. The peripheral labyrinthine stimulation sets into activity a machinery which gives rise to nystagmus in the same way as the spinal cord sets up the scratch reflex as a response to a stimulus produced in the skin. Several experiments have led to the conclusions that the production of the rhythm of the nystagmus, i. e., of the quick component, needs more neurons than those used in the production of the slow component. These additional neurons lie in the reticular substance. Developing a theory advanced by Bárány in 1907, a diagram explaining the production of the rhythm of the nystagmus is offered (fig. 27). This diagram can possibly explain the production of other rhythmic reflexes.

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42. Leidler, R.: Experimentelle Untersuchungen über das Endigungsgebiet des Nerven octavus II, Arb. a. d. neurol. Inst. a. d. Wien. Univ. **21**:151, 1914, Experiment XVII.

43. Spiegel, E. A., and Sato, G.: Experimental Studien am Nervensystem: Ueber den Erregungszustand der medullären Zentren nach doppelseitiger Labyrinthausschaltung, Arch. f. d. ges. Physiol. **215**:106, 1926.

44. Brown, T. G.: Studies in the Physiology of the Nervous System: VIII. Neural Balance and Reflex Reversal, Quart. J. Exper. Physiol. **4**:273, 1911. Beritoff: Allgemeine Characteristik der Tätigkeit des Nervensystems, Ergebn. d. Physiol. **20**:407, 1922. Lorente de Nó.[12b]

The reciprocal innervation of muscles is conditioned because only the motoneurons of one muscle of the pair receive excitatory impulses; the motoneurons of the antagonistic muscle come to a state of rest. No indication of active inhibition of motoneurons has been found in cases in which inhibition has been demonstrated; the process of inhibition took place in internuncial relays; however, no assumption can be made as to the intimate nature of inhibition. When the internuncial relays or simply secondary pathways are destroyed, antagonistic muscles may

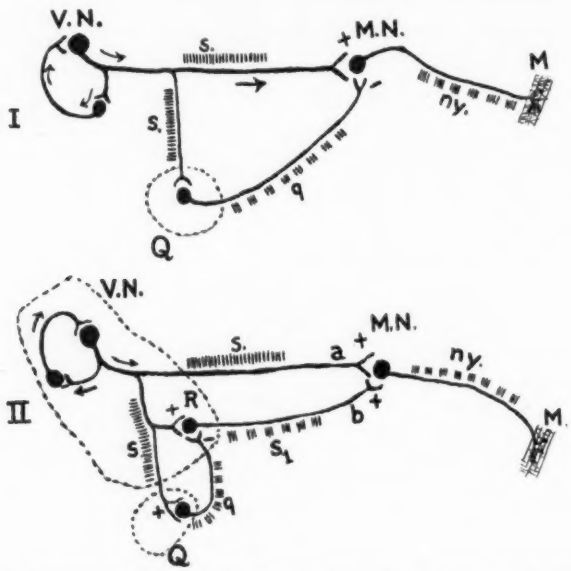

Fig. 27.—Diagram explaining the production of the rhythm of the nystagmus. *I*, vestibular nuclei (*V.n.*) (including reticular relays) set up a continuous stream of impulses *s*, which on the one hand excite the motoneuron *M* and on the other hand excite the system of neurons *Q*, which set up a discontinuous series of inhibitory impulses (*q*). When the *s* impulses alone arrive at *M.N.*, the muscle contracts; when *s* and *q* arrive at the same time they cancel each other, the motoneuron is not excited and the muscle relaxes. This diagram, however, is not very likely because it does not consider internuncial neurons in the system *V.N.* In *II*, *V.N.* includes internuncial neurons *R* and explains the production of the rhythm without inhibition of motoneurons. On the motoneuron *M* end two paths *a* and *b*; *a* is conducting a continuous series of excitatory impulses, which, however, are unable to set up a motor discharge unless fiber *b* is also discharging; but fiber *b*, as a consequence of the activity of system *Q*, is conducting a discontinuous series of discharges of cells *R*, which is thought of being under the same circumstances as cell *M.N.* in *I*. *II* is in agreement with the results of all the experiments; the quick component of the nystagmus will disappear when either the afferent or the efferent paths of *Q* are severed. This diagram must be completed with recurrent fibers (fig. 3, *II*, *III*) from *Q* to *R* and from *R* to *V.N.* The system *R-Q* must be composed of numerous cells; otherwise its long latent period could not be explained.

show co-contraction, which again is changed into reciprocal innervation when still more internuncial pathways are destroyed.

The uncrossed as well as the crossed secondary vestibular paths carry excitatory impulses.

In the vestibular system there is no "localization" of reflexes in anatomic nuclei; the whole system is a functional unit; reflex reactions may be produced as long as the reflex arc is closed through one of the multiple pathways; on the other hand, lesions in any part of the vestibular system modify all the reflexes.

CORTICIFUGAL PATHWAYS FOR MASTICATION, LAPPING AND OTHER MOTOR FUNCTIONS IN THE CAT

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It has long been known that stimulation of the appropriate part of the cerebral cortex will give rise to rhythmic, serially repeated movements of mastication. As a result of early experimental work the existence of an intercalary subcortical center was postulated in the reaction. This center was thought to receive the impulses from the cortex and transform them into the rhythmic series which bring about the movements involved in eating. Evidence both from electrical stimulation and from experiments in which degeneration was employed pointed to the substantia nigra as being such a center.

The corticifugal pathway for reaction of mastication was first traced through the internal capsule by Réthi [1] in the rabbit. By sectioning the brain and stimulating the exposed end with faradic current he obtained rhythmic masticatory movements as far caudally as the subthalamic region; below that level he obtained only a steady closure of the jaws. He postulated an intercalary center in either the diencephalon or the upper part of the midbrain but did not specifically designate the substantia nigra as such.

Bechterew [2] and his students obtained occasional movements related to the taking of food and chewing from electrical stimulation of the lateral part of the substantia nigra in dogs and, in connection with similar results related to swallowing, assumed the existence of a special center for swallowing and chewing in the substantia nigra.

The anatomic basis for this point of view was furnished by Economo [3] in the rabbit and cat. In Marchi material, after lesion of

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1. Réthi, L.: Das Rindenfeld, die subcorticalen Bahnen und das Coordinationcentrum des Kauens und Schluckens, Sitzungsb. d. k. Akad. d. Wissensch. Math.-naturw. Cl., Wien **102**:359, 1893.

2. Bechterew, W.: Die Funktionen der Nervencentra, Jena, Gustav Fischer, 1909, vol. 2.

3. Economo, C. J.: Die centralen Bahnen des Kau- und Schluckactes, Arch. f. d. ges. Physiol. **91**:629, 1902.

the cortical area for mastication, he was able to trace degenerating fibers back to their endings in the medial portion of the substantia nigra.

While no one has repeated the work with the degeneration method, the results from electric stimulation have been reinvestigated by Miller [4] and by us. Miller, by sectioning the brain of rabbits and stimulating the exposed end, found that rhythmic mastication gave way to steady closure of the jaws between the levels of the mamillary bodies and the substantia nigra. He recognized that closure of the jaws resulted from the spread of current to the fifth nerve and explained his inability to obtain the reaction of mastication below the level of the mamillary bodies on the ground that the corticifugal fibers for mastication here left the peduncle and coursed as scattered, aberrant fibers to their respective motor nuclei. Since rhythmic mastication had not been followed below the caudal end of the diencephalon, i. e., up to the site of the substantia nigra, it seemed that one interpretation was as valid as another, and we decided to investigate the question with the improved technic made possible by the use of the Horsley-Clarke apparatus.

The results obtained show that rhythmic movements of the mouth and tongue, which usually make their appearance as lapping in the cat, may be followed back from the cortex in the internal capsule and cerebral peduncle, ventral to the substantia nigra, as far as the caudal end of the pons. After destruction of the cortical area for mastication and degeneration of the corticifugal pathway no trace of any such reactions could be obtained by stimulation of the substantia nigra or of any other center in the brain stem.

METHODS

Data were obtained from experiments on forty-one cats utilized in the following ways: The cortical areas for chewing and lapping were demarcated on the surface of the cortex in three cats; these reactions were followed back in the internal capsule to the anterior end of the diencephalon in seven cats; their location in the diencephalon and upper part of the midbrain was determined in twenty-two cats, and their position in the midbrain and pons in six cats. The anterior third of one cerebral hemisphere was removed in each of three cats, and after sufficient time had elapsed for degeneration of the corticifugal fibers the brain stem was thoroughly stimulated from the anterior diencephalon down into the pons.

The large number of cats used for the experiments performed on the diencephalon and upper part of the midbrain should not be taken to indicate difficulty in obtaining responses at these levels. The data accumulated during another series of experiments by two of us (Ranson and Magoun [5]) and are reported here for the first time.

4. Miller, F. R.: The Cortical Paths for Mastication and Deglutition, J. Physiol. **53**:473, 1920.

5. Ranson, S. W., and Magoun, H.: Respiratory and Pupillary Reactions Induced by Electrical Stimulation of the Hypothalamus, Arch. Neurol. & Psychiat. **29**:1179 (June) 1933.

For removal of the anterior portion of the hemisphere, the cat was put under deep pentobarbital sodium anesthesia, 40 mg. per kilogram. All aseptic precautions were taken. The left temporal muscle was reflected, and a section of the left side of the calvarium three fourths of an inch wide was removed from the midline to the base of the cranium. The posterior margin of this opening was approximately on a line with the anterior ectosylvian sulcus. The left hemisphere was sectioned in the frontal plane at the level of this sulcus, and the anterior end was removed. The cavity was allowed to fill with clotted blood, and after bleeding had ceased the wound was closed. After a postoperative period of three weeks in one cat and a month and a half in the other two both sides of the brain stem were stimulated by means of the Horsley-Clarke apparatus.

In all the experiments in which stimulation was applied the cats were under light pentobarbital sodium anesthesia of from 16 to 19 mg. per kilogram; supplemental ether was added, if necessary, by means of a tracheal cannula and ether bottle.

In preparation for stimulating the surface of the cortex in the three normal cats the left temporal muscle was reflected and partly cut away, the calvarium was freely removed, and the dura was divided. The left hemisphere was tilted with a spatula to permit access to its ventral surface, and the region of the anterior sylvian gyrus was stimulated with a bipolar platinum electrode the tips of which were 1.5 mm. apart. The current was obtained from a single dry cell registering 1.5 amperes connected to a Harvard inductorium set at 8 cm.

In all the other experiments in which stimulation was applied the Horsley-Clarke apparatus was employed. The experiments were usually conducted on the left side of the brain. The temporal muscle was reflected, and about 1 square inch of the calvarium was removed, after which the apparatus was adjusted to the cat's head. The instrument and its manner of use have been described by Ingram, Ranson, Hannett, Zeiss and Terwilliger.[6] It may be described briefly as an apparatus accurately adjustable to the animal's head, which enables a bipolar needle electrode to be oriented in the brain by readings along the anteroposterior, mediolateral and dorsoventral scales of the instrument.

The bipolar needle electrode used in our experiments consisted of two lengths of nichrome wire insulated and cemented together with baked enamel. Its widest diameter was 0.8 mm., and the shortest distance between the exposed tips was about 0.2 mm. The current was supplied by a single dry cell registering 1.5 amperes attached to a Harvard inductorium in which the secondary coil was set at either 8 or 9 cm. The electrode was inserted vertically through the dura into the brain at a point 1 mm. from the midline. After stimuli had been applied it was removed and reinserted 1 mm. farther laterally. Five or more such punctures were made in one transverse plane with millimeter intervals between the punctures; then the electrode was moved 2 mm. caudally, and another similar transverse row of punctures was completed. As many as twenty rows were sometimes made in a single brain, but usually the number was only five or six, the process being rather time-consuming. Stimulation was applied at millimeter intervals along each of the punctures as the needle was inserted through the brain from the dorsal part of the cortex to the base. In the telencephalon the area stimulated had its most dorsal extent on a level with the cruciate sulcus, while farther caudally practically the whole depth of the brain stem was explored. The coordinates of each point stimu-

6. Ingram, W. R.; Ranson, S. W.; Hannett, F. I.; Zeiss, F. R., and Terwilliger, E. H.: Results of Stimulation of the Tegmentum with the Horsley-Clarke Stereotaxic Apparatus, Arch. Neurol. & Psychiat. **28**:513 (Sept.) 1932.

lated, together with the reactions observed, were recorded in systematic notes and were later correlated with microscopic sections of the brain.

At the conclusion of each experiment four wires were inserted into the brain with the electrode carrier; these served as guides in cutting out the block for microscopic study and made it possible to cut the sections in the same planes as those occupied by the punctures. An injection of a solution of formaldehyde was made into the head and the brain was removed, trimmed and prepared according to the Weil method.

The rows of punctures were identified in the sections, the punctures appearing as fine vertical tracks of hemorrhage. Drawings were made of the level of each row with the Edinger projection apparatus. The shrinkage of the brain due to embedding was calculated and taken into account in each instance, and the location of the stimulated points was determined by measuring from the base of each puncture. These locations could be checked by comparison with the position of points which had been definitely marked by stopping certain of the punctures when an especially marked reaction was obtained. Such points could be readily identified at the ends of the punctures. All the points stimulated were plotted on the drawings, and their corresponding reactions were differentiated by means of colored crayons and symbols. In this way 115 drawings of cross-sections of the brain were obtained, in which the locations of all the points stimulated, together with the reactions obtained, were indicated. The arrangement of the large amount of data in this way makes for easy study, correlation and reference.

MASTICATION AREA ON THE SURFACE OF THE CORTEX

The demarcation of the electrically excitable field for mastication on the surface of the cortex was undertaken in order to check its location with the results obtained by Ferrier,[7] Economo,[3] Sherrington[8] and Bremer,[9] all of whom worked with the cat. The portion of the hemisphere from which the response of mastication is obtained was not explored by Stout,[10] as he did not regard this region as part of the "motor area." The attention of other recent workers on the cerebral cortex of the cat has been directed only toward the sigmoid gyri, together with the area frontalis. The comparatively inaccessible position of the area for mastication may explain why it has been omitted from consideration in recent literature.

Rhythmic chewing and lapping may be obtained from stimulation of the rostral end of the anterior sylvian gyrus and the adjacent portion of the rhinal fissure (fig. 1). While the fields for the two reactions intergrade, purer chewing is observed from the anterior sylvian gyrus

7. Ferrier, D.: The Function of the Brain, ed. 2, London, Smith, Elder & Co., 1886, vol. 23.

8. Sherrington, C. S.: Reflexes Excitable in the Cat from Pinna, Vibrissae and Jaws, J. Physiol. **51**:404, 1917.

9. Bremer, F.: Physiologie nerveuse de la mastication chez le chat et le lapin, Arch. internat. de physiol. **21**:308, 1923.

10. Stout, J. D.: On the Motor Functions of the Cerebral Cortex of the Cat, Psychobiol. **1**:177, 1917.

(fig. 1, lined area), and purer lapping from the rhinal fissure; in one cat lapping was obtained from the olfactory tract (fig. 1, solid black).

STIMULATION OF THE INTERIOR OF THE BRAIN

While the initial interest in the experiments lay in the movements of mastication, data on other motor functions accumulated. No attempt was made to explore the motor cortex systematically, but some reasonably precise information was obtained regarding the course of various corticifugal motor fibers in the white matter of the hemisphere, internal capsule and basis pedunculi.

The method of graphic illustration seems to be the most satisfactory way of presenting the accuracy of localization which is possible with the use of the Horsley-Clarke apparatus. As it is impossible to present the

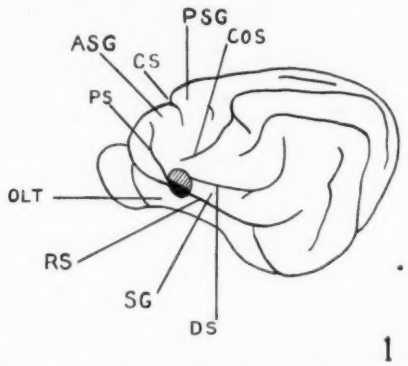

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Fig. 1.—Lateral view of the left cerebral hemisphere of the cat. The portion of the anterior sylvian gyrus stimulation of which yields the response of chewing is indicated by the lined area; the adjacent region of the rhinal sulcus, from which the response of lapping is obtained, is shown by the black area. The abbreviations have the following meanings: *ASG*, anterior sigmoid gyrus; *COS*, coronal sulcus; *CS*, cruciate sulcus; *DS*, diagonal sulcus; *OLT*, olfactory tract; *PS*, presylvian sulcus; *PSG*, postsigmoid gyrus; *RS*, rhinal sulcus, and *SG*, anterior sylvian gyrus. This figure is based on data obtained from experiments performed on three cats.

large amount of data in its totality, twelve representative levels have been selected. These extend from the motor cortex back to the caudal end of the pons. Since the range of exploration in any one cat covered only a portion of this distance, it has been necessary to take different levels from different cats. Except for the figures illustrating the experiments on the diencephalon and upper part of the midbrain, each figure represents data based on experiments made on a single cat. We wish to make it clear, however, that the data indicated at any one level are abundantly corroborated and verified by similar data obtained from adjacent levels in other cats.

The typical reactions obtained may first be described. Rhythmic movements of the mouth and tongue were elicited usually as lapping. This resembled in every particular the lapping of a drinking cat, except that the tongue usually deviated to the contralateral side. Chewing was obtained occasionally. The predominance of the lapping may indicate a greater cortical representation. Simple movements of the mouth or tongue were never observed. Movements of the face and neck were noted rarely.

The responses of the forelimb were usually rhythmically repeated and were always contralateral. The movements characteristically resembled stepping but sometimes suggested digging and at other times striking. Occasionally, in the basis pedunculi, lapping and stepping were obtained together. At such times they appeared synchronized: the leg went forward as the tongue was protruded. The nonrhythmic or simple responses of the forelimb were always those of flexion, with the exception of two instances in which extension occurred. Rostral to the internal capsule the simple reactions of the forelimb were almost always associated with similar reactions of the hindlimb. No movements of the trunk were ever observed.

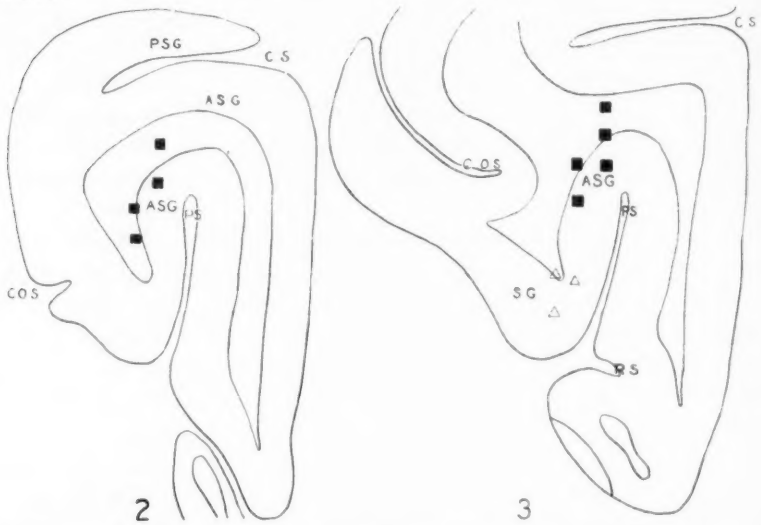
The responses of the hindlimb were usually those of simple flexion, but occasionally they were rhythmically repeated and resembled stepping. They were always contralateral, with one exception in which ipsilateral stepping of the hindlimb was associated with contralateral stepping of the forelimb. This might be interpreted as the result of a spinal mechanism linking up with the cortical impulse. Tail movements were found rarely.

Telencephalon.—The only response obtained from the most rostral stimuli was that of rhythmic movements of the contralateral forelimb (fig. 2, black squares). This is in keeping with the rostral surface location of response of the forelimb on the anterior sigmoid gyrus. It is of interest that the response is obtained from the buried portion of the anterior sigmoid gyrus, which forms one wall of the deep presylvian sulcus. There is by no means a sufficient amount of data to show that the dorsal or superficial part of the gyrus is not responsive, but rhythmic responses of the forelimb do arise from its ventral or deep portion and, farther caudally, from the white matter adjacent to it.

As these responses of the forelimb are traced farther caudally to the level represented in figure 3, they are seen to have shifted dorsomedially to a position in the white matter about on a line with the dorsal end of the presylvian sulcus, and, in the main, close to the cortex which this sulcus indents. As will be seen in the later, progressively more caudal figures, the rhythmic reactions of the forelimb remain in approximately this same position until the internal capsule begins to form.

At this same level, the most rostral responses of chewing appear (fig. 3, white triangles). The effective points are seen to lie among the fibers derived from the ventromedial portion of the anterior sylvian gyrus, their position being more medial than that indicated by stimulation of the surface.

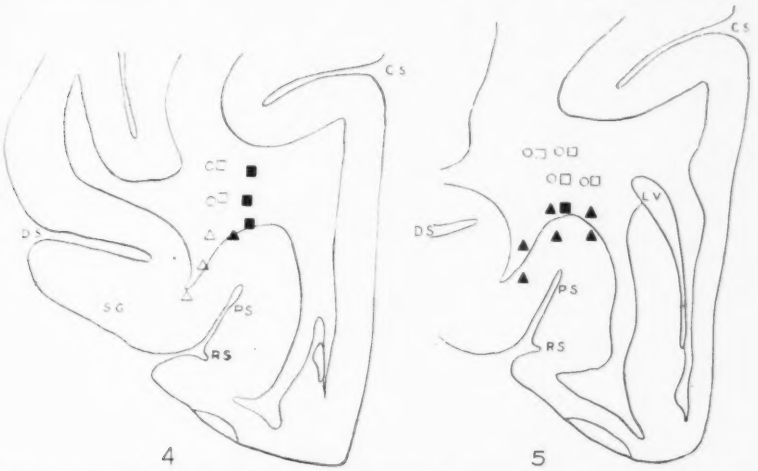
Passing caudally to figure 4, one finds that the responses of chewing have shifted somewhat dorsally in the white matter. The location of the first response of lapping is indicated (black triangle). The more rostral position of the responses of chewing, as compared with that of lapping, reveals another instance in which stimulation of the interior of the cortex yields information which is not apparent from stimulation of its surface.



Figs. 2 and 3.—Cross-sections of left side of brain, showing level through rostral tip of posterior sigmoid gyrus and the successively caudal level. Each symbol represents the location of a point stimulation of which yields the movement specified. The black squares indicate rhythmic stepping of the contralateral forelimb; the white triangles, rhythmic chewing. The letters have the following indications: *ASG,* anterior sigmoid gyrus; *COS,* coronal sulcus; *CS,* cruciate sulcus; *PS,* presylvian sulcus; *PSG,* postsigmoid sulcus; *RS,* rhinal sulcus, and *SG,* anterior sylvian gyrus.

Rhythmic movements of the forelimb (black squares) are obtained at the level represented in figure 4 from about the position already described, and the first simple flexions of the forelimb (white squares) associated with simple flexions of the hindlimb (empty circles) are elicited dorsolaterally in the broadening band of white matter which will form the medial portion of the internal capsule.

The main body of the responses of rhythmic lapping first appear at the level indicated in figure 5. They are seen here (black triangles) to form an arc in the cortex and the adjacent white matter around the end of the presylvian sulcus and along its lateral surface. The area of cortex from which the lapping is derived appears to be larger than that for chewing (compare figs. 3 and 4, white triangles). This may account for the greater representation of lapping over chewing which obtains from the level of figure 5 caudally. Once the lapping has come in it is only rarely that a response of chewing is found. The few responses of chewing that did occur occupied approximately the same position that lapping would otherwise hold. The situation of the responses of lapping in the cortex lateral and dorsal to the presylvian



Figs. 4 and 5.—Successively caudal levels through the left hemisphere. The symbols have the following meanings: black squares, rhythmic stepping of the contralateral forelimb; white squares, simple flexions of the contralateral forelimb; white circles, simple flexion of the contralateral hindlimb; black triangles, rhythmic lapping, and white triangles, rhythmic chewing. *CS* indicates the cruciate sulcus; *DS*, the diagonal sulcus; *LV*, the lateral ventricle; *PS*, the presylvian sulcus; *RS*, the rhinal sulcus, and *SG*, the anterior sylvian gyrus.

sulcus explains their appearance from the rhinal fissure (into which the presylvian sulcus opens ventrally) in stimulation of the surface of the hemisphere.

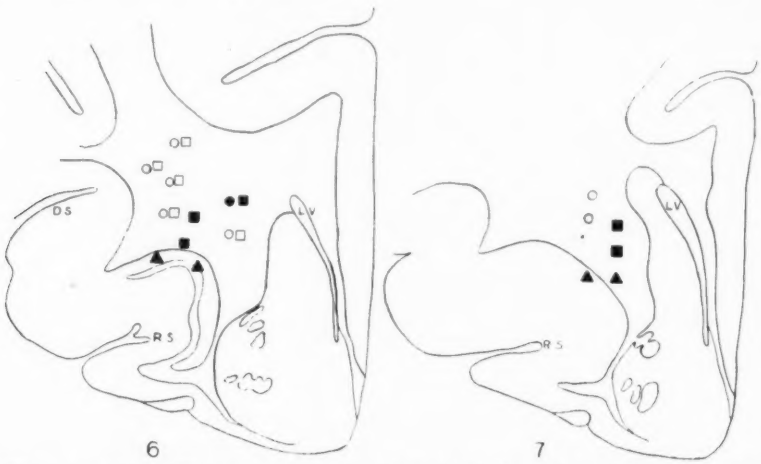
At the level represented in figure 5, rhythmic movements of the forelimb retain their position in the white matter opposite the end of the presylvian sulcus. Simple flexion of the forelimb and hindlimb is more strongly represented here.

At a level just rostral to the internal capsule (fig. 6) the responses of lapping and of rhythmic movements of the forelimb have shifted

somewhat medially, while those for simple movements of the forelimb and hindlimb retain about the same position.

It is not, however, until a level is reached at which the internal capsule begins to form (fig. 7) that the reactions have shifted medially enough to give an idea as to what their arrangement in the capsule will be. The area for lapping occupies a ventral position, that for rhythmic movements of the forelimb occurs dorsal to it, and that for responses of the hindlimb is the most dorsally situated.

This laminate arrangement is more fully indicated in figure 8, which represents the level at which the medial or responsive portion of the internal capsule is completely formed. The most medial portion of the capsule is negative. Next, in a ventromedial position, are situated the responses of lapping; dorsal to these, in an intermediate



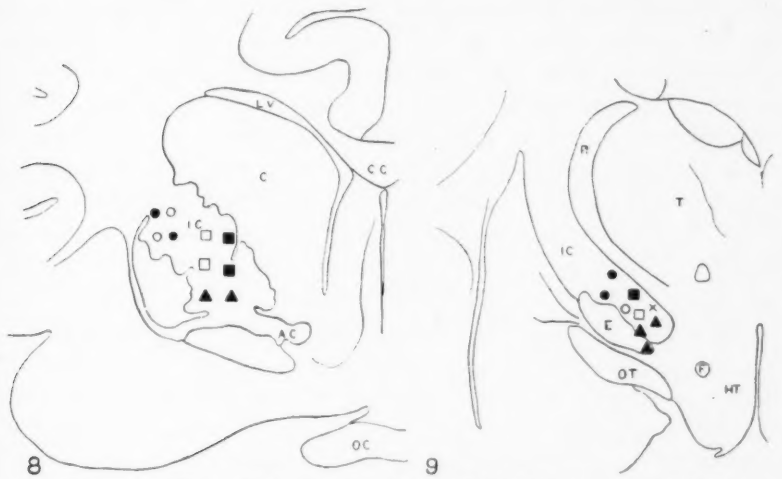
Figs. 6 and 7.—Figure 6 shows the first indication of the formation of the internal capsule; figure 7 shows the level at which its size has increased. The black squares represent rhythmic stepping of the contralateral forelimb; the white squares, simple flexion of the contralateral forelimb; the black circles, rhythmic stepping of the contralateral hindlimb; the white circles, simple flexion of the contralateral hindlimb, and the black triangles, rhythmic lapping. *DS* indicates the diagonal sulcus; *LV*, the lateral ventricle, and *RS*, the rhinal sulcus.

location, are the rhythmic and simple responses of the forelimb, while most laterally and dorsally the reactions of the hindlimb are found. In the few cats in which facial movements occurred these reactions lay close to the responses of lapping.

There is, then, a ventromedian-dorsolateral distribution of motor functions in the internal capsule which corresponds to the antero-posterior distribution of motor activity along the body of the animal. The same holds true for the cerebral peduncle, as will be seen. An identical distribution in the cerebral peduncle of the orang-utan was

pointed out by Leyton and Sherrington.[11] It is of great interest that an animal like the cat, in which the arrangement of functions on the surface of the cortex does not fall into any orderly distribution along a precentral gyrus, nevertheless shows, in its internal capsule and basis pedunculi, an arrangement of functions of the head, forelimb and hindlimb, from the medial portion laterad, analogous to that found in anthropoids.

Diencephalon.—Because of the large number of cats used for the experiments on the diencephalon and upper part of the midbrain the planes of stimulation coincided in several cats. Therefore the sum of the data from the coinciding level is considered in the study of this



Figs. 8 and 9.—Figure 8 shows the medial, well developed portion of the internal capsule, the anterior commissure and the optic chiasm; figure 9 represents a level through the middle of the diencephalon. The black squares indicate rhythmic stepping of the contralateral forelimb; the white squares, simple flexion of the contralateral forelimb; the black circles, rhythmic stepping of the contralateral hindlimb; the white circles, simple flexion of the contralateral hindlimb, and the black triangles, rhythmic lapping; the cross represents movements of the face. The letters have the following indications: *AC*, anterior commissure; *C*, caudate nucleus; *CC*, corpus callosum; *E*, entopeduncular nucleus; *F*, fornix; *HT*, hypothalamus; *IC*, internal capsule; *LV*, lateral ventricle; *OC*, optic chiasm; *OT*, optic tract; *R*, reticular nucleus, and *T*, thalamus. Figure 9 represents the data from experiments performed on five cats.

region. The drawings of the other levels shown represent the data from only one cat. At the risk of possible confusion the data from

11. Leyton, A. S. F., and Sherrington, C. S.: Observations on the Excitable Cortex of the Chimpanzee, Orang-Utan and Gorilla, Quart. J. Exper. Physiol. **2**: 135, 1917.

several cats are shown in each of figures 9, 10, 11 and 12. This is done in order to give an idea of how the results obtained from the experiments on different cats correspond and may be combined to form a uniform picture.

The level through the caudal portion of the internal capsule (fig. 9) shows the motor responses located near its medial border in the same order as has been previously indicated. Most medially situated are the responses of lapping. While the majority of these are located in the internal capsule proper, occasional responses are elicited in the entopeduncular nucleus. It is suggested that these responses represent excitation of the adjacent fibers of the capsule or of the corticifugal fibers which stream through the nucleus in numerous clusters. There is no evidence that the entopeduncular nucleus modifies or bears any relation to the response of lapping. Responses of the forelimb at this level occupy an intermediate position between the medially situated reactions of lapping and the laterally situated movements of the hindlimb.

As the caudal end of the internal capsule becomes the rostral end of the basis pedunculi (figs. 10 and 11), a rearrangement of the motor responses begins to take place. It is apparent that a clumping together of the responses occurs, which is augmented by the shifting of the response of lapping from their medial position to a more lateral one close to the dorsal surface of the basis pedunculi.

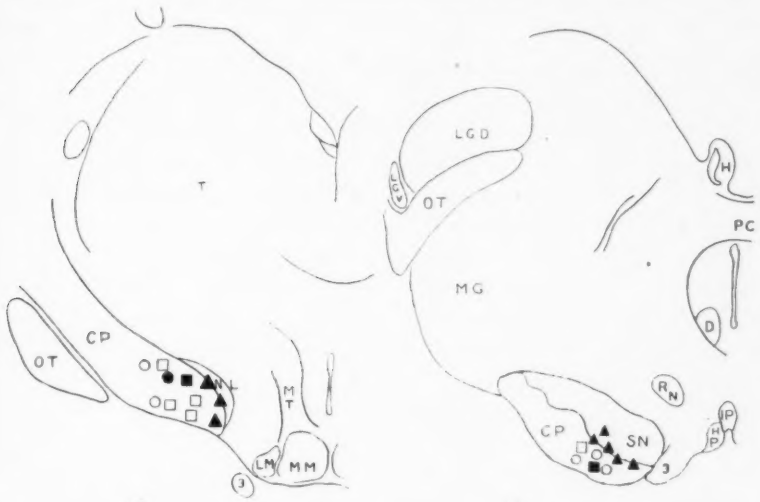
Midbrain.—This rearrangement of functional pathways is more fully indicated at the level of the substantia nigra, where the cerebral peduncle is well formed (fig. 11). The responses of the limbs appear gathered into a bundle slightly medial to the middle of the basis pedunculi, while those of lapping lie along its dorsal surface and are only occasionally found near the medial end.

The distribution of the responses of lapping at this level (fig. 11) is of great interest in consideration of the masticatory function ascribed to the substantia nigra. Typical reactions of lapping were obtained fifteen times from the level of the mesencephalon in the total number of cats stimulated in this area. Of these fifteen responses, two arose from the substantia nigra close to the pyramidal fibers, eight were obtained from the basis pedunculi after an immediately previous stimulus in the substantia nigra had given negative results, and five were obtained from the dorsal surface of the basis pedunculi after a similar stimulus in the substantia nigra had given negative results. In all, twenty-two stimuli of the substantia nigra gave negative results. This distribution of the reactions clearly seems to indicate that the corticifugal pathway for lapping bears no relation to the substantia nigra other than to lie rather near it. In order, however, to check the possibility of a relay here, the substantia nigra was thoroughly stimulated after degeneration

of the corticifugal elements. The negative results of these experiments will be indicated later.

Pons.—As the pyramidal fibers begin to swing medially into the rostral pons, all the motor responses are situated a little medially to the middle of the basis pedunculi (fig. 12). While the reactions of the forelimb and hindlimb are bunched together, the responses of the forelimb are the more medial of the two. Lapping is obtained from the same region but a little more dorsally than the other responses.

As the cortical motor fibers continue to swing medially, the responses of lapping become situated more and more laterally in their midst. The last response of lapping was obtained in the extreme caudal end of the



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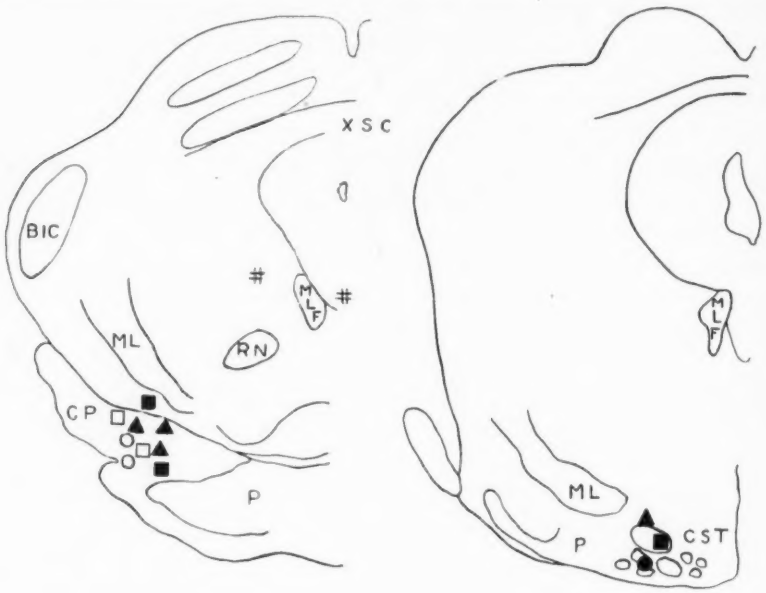
Figs. 10 and 11.—Figure 10 shows a level through the caudal end of the diencephalon; figure 11 shows the anterior end of the midbrain. The black squares indicate rhythmic stepping of the contralateral forelimb; the white squares, simple flexion of the contralateral forelimb; the black circle, rhythmic stepping of the contralateral hindlimb; the white circles, simple flexion of the contralateral hindlimb, and the black triangles, rhythmic lapping. *CP* indicates the cerebral peduncle, basis pedunculi; *D*, the nucleus of Darkschewitsch; *H*, the habenula; *HP*, the habenulopeduncular tract; *IP*, the interpeduncular nucleus; *LGD*, the dorsal nucleus of the lateral geniculate body; *LGV*, the ventral nucleus of the lateral geniculate body; *LM*, the lateral mamillary nucleus; *MG*, the medial geniculate body; *MM*, the medial mamillary nucleus; *MT*, the mamillothalamic tract; *NL*, the nucleus of Luys; *OT*, the optic tract; *PC*, the posterior commissure; *RN*, the red nucleus; *SN*, the substantia nigra, and *T*, the thalamus. The numeral *3* indicates the oculomotor nerve. Figure 10 represents the data obtained from experiments on six cats; figure 11, the data from experiments on seven cats.

pons on the dorsolateral surface of the bundles of cortical fibers, immediately rostral to the formation of the pyramid (fig. 13). This

point is a trifle over 2 mm. in front of the fifth motor nucleus, stimulation of which yields only a steady closure of the jaws. It is suggested that in these 2 mm. the fibers concerned with lapping ascend through the reticular formation to the region of the fifth motor nucleus in clusters too scattered to be activated by a localized stimulus.

The present series throws little light on the cortical component running back to the hypoglossal nucleus. The medulla caudal to the fifth motor nucleus was not thoroughly explored, but the stimuli that were applied there yielded no clearcut evidence. Hypoglossal reactions consisting of nonrhythmic opening of the mouth and protrusion of the



12 13

Figs. 12 and 13.—Cross-sections showing levels at rostral portion of pons and at extreme caudal end of pons. The significance of the symbols and abbreviations is as follows: black squares, rhythmic stepping of the contralateral forelimb; white squares, simple flexion of the contralateral forelimb; black circles, rhythmic stepping of the contralateral hindlimb; white circles, simple flexion of the contralateral hindlimb, and black triangles, rhythmic lapping. The number sign indicates simple opening of the mouth and protrusion of the tongue. *BIC* indicates brachium of the inferior colliculus; *CP*, the cerebral peduncle, basic pedunculi; *CST*, the corticospinal tract; *ML*, the medial lemniscus; *MLF*, the median longitudinal fasciculus; *P*, the pons; *RN*, the red nucleus, and *XSC*, the commissure of the superior colliculus. Figure 12 represents the data from experiments performed on four cats.

tongue to the contralateral side have been obtained from stimuli applied not far from the medial longitudinal fasciculus in the midbrain (fig. 12).

It is, of course, a question whether these represent the reactions of aberrant cortical fibers. Furthermore, since the reaction of rhythmic lapping can be followed down to the caudal end of the pons in the pyramidal tract, no relation between the two reactions is apparent.

Stimulation of the Substantia Nigra After Degeneration of the Pyramidal Tract.—In order to check the possibility of an intercalary center in the corticifugal pathway for mastication, the brain stem was thoroughly stimulated after removal of the anterior third of one cerebral hemisphere in each of three cats. The time allowed for degeneration was three weeks in one cat and a month and a half in the other two. In the first cat stimulation of one point in the dorsomedial portion of the basis pedunculi at the level of the rostral end of the substantia nigra on the side on which the operation had been performed yielded irregular lapping movements. These movements were long delayed and were very haltingly performed. The most probable explanation of their occurrence seems to be that a small amount of the cortex of the pathway for lapping was left intact at operation. Stimulation in the other two cats gave entirely negative results on the side on which the anterior third of the cerebral hemisphere had been removed. In the three cats taken together, twenty-three points were stimulated in the substantia nigra on the side of the brain on which the operation had been performed and were distributed in such a manner as to cover both its rostrocaudal and its mediolateral extent. Stimulation of all these points gave negative results.

Should a relay in the path for mastication take place at the substantia nigra, the secondary element must travel back in the basis pedunculi, as was previously shown in the normal cats. Stimuli applied caudal to the substantia nigra are significant, then, in demonstrating the absence of such a secondary element. In all three cats, stimuli applied along six transverse rows on the side of the brain on which the operation had been performed thoroughly covered the brain stem between the caudal end of the substantia nigra and the middle of the pons. The entire cerebral peduncle was repeatedly explored in these rows. All these stimuli gave negative results.

On the opposite, normal side of the brain in the two cats which survived for one and one-half months the typical response of lapping was readily followed, in the basis pedunculi, from the diencephalon to the middle of the pons. In the other cat of this series the normal side was not thoroughly explored.

The evidence from these three animals concurs with that from normal cats to show that the corticifugal function of mastication follows an uninterrupted path from the cortex back to the region of the fifth motor nucleus, and clearly indicates that the substantia nigra cannot be regarded as an intercalary center in its course.

COMMENT

The rhythmic, serially repeated character of certain cortical responses merits some attention. That rhythmic responses may be obtained from cortical stimulation is an ascertained fact. Vogt and Vogt [12] obtained rhythmic movements of mastication from stimulation of the cortex of representative marsupials, insectivores and primates. Leyton and Sherrington [11] obtained them in anthropoids. Ferrier,[7] Economo,[3] Sherrington,[8] Bremer [9] and we ourselves have obtained rhythmic mastication from cortical stimulation in the cat. Rhythmic movements of the limbs do not appear to be so general, but Sherrington,[8] Weed and Langworthy,[13] Langworthy,[14] and we ourselves have observed them from stimulation of the cortex of the cat.

If rhythmic responses were induced by cortical stimulation alone, one might conclude that they were dependent on intrinsic cortical associations. It is impossible, however, to explain the rhythmic responses obtained from stimulation of the internal capsule and the cerebral peduncle on these grounds. We should suggest that a series of cortical impulses, either excitatory alone, or a combination of excitatory and inhibitory similar to those obtained by Cooper and Denny-Brown,[15] sets into play a complicated rhythmic bulbar or spinal center. Such an interpretation is in keeping with the work of Bremer,[9] who obtained rhythmic movements of mastication from mechanical stimulation of the mucosa of the buccal commissure in pontile cats. The inherent rhythmicity of the spinal motor centers concerned in stepping was emphasized by Graham Brown.[16]

While only incidental information is furnished by the present series of experiments on the demarcation of the cortical motor area of the cat, the large number of responses obtained from the buried cortex forming the lateral and dorsal wall of the presylvian sulcus is of interest. McKibben and Wheelis [17] emphasized the importance of taking the buried cortex into consideration in mapping the cortical motor area of the cat. We support this emphasis and call attention to the buried cortex lateral and dorsal to the presylvian sulcus.

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SUMMARY

The data bearing on the localization of the corticifugal pathways for responses of the forelimbs and hindlimbs and for those of lapping may now be brought together. At least a portion of the rhythmic responses of the forelimbs arise from the buried or deep part of the anterior sigmoid gyrus adjacent to the presylvian sulcus (fig. 2). They shift dorsomedially to a position in the white matter opposite the end of the presylvian sulcus and, in the main, close to the cortex which this sulcus indents (fig. 3). This position is retained back to the point of formation of the internal capsule (figs. 4, 5 and 6). Here the responses shift medially and somewhat ventrally to occupy an intermediate position in the internal capsule between the responses of lapping ventromedially and the responses of the hindlimb dorsolaterally (figs. 7 and 8).

Rhythmic movements of chewing arise from the medial portion of the rostral end of the anterior sylvian gyrus (fig. 3). They begin to shift dorsomedially at the point where the responses of lapping appear (fig. 4), and are only occasionally found below this level. It is suggested that a greater cortical representation of lapping enables it to predominate over chewing. When chewing movements do occur they arise from approximately the same area from which lapping would otherwise be obtained.

Rhythmic lapping movements arise from the cortex around the end of the presylvian sulcus and along its lateral surface (figs. 4 and 5). As the lapping reactions are traced caudally they swing medially to form a kind of arc along the edge of the adjacent white matter under the responses of the limbs (fig. 6) and enter the ventral part of the forming internal capsule (fig. 7). They come to lie in the ventromedial portion of the internal capsule, with a small unresponsive area medial to them, and with the field for the responses of the forelimb dorsolaterally situated (fig. 8).

Simple flexions of the forelimb and hindlimb make their first appearance caudal to the first rhythmic responses of the forelimb in the dorsolateral part of the broad band of white matter between the cruciate and presylvian sulci (figs. 4 and 5). It is from this band of white matter that the medial, responsive part of the internal capsule is formed, and as this transformation takes place, the simple responses of the hindlimb retain their position relative to those of the other functions (figs. 6 and 7). As a result the movements of the hindlimb come to occupy a dorsolateral position in the medial, responsive portion of the internal capsule (fig. 8). As they enter the internal capsule, the simple responses of the forelimb shift ventromedially to the position occupied by the other movements of the forelimb in the intermediate position already described (fig. 8).

The same distribution of responses which obtained in the internal capsule—medial for the head, intermediate for the forelimb and lateral for the hindlimb—is found in the rostral portion of the basis pedunculi (fig. 10). Farther caudally, however, a rearrangement of these reactions takes place. The movements of the limbs cluster together slightly medial to the middle of the basis pedunculi, the responses of the forelimb being usually on the medial side of this cluster. Concomitantly, the reactions of lapping shift dorsolaterally from their former medial position to a position close to the dorsal surface of the basis pedunculi, in the main, on a line with the other responses (figs. 11 and 12).

As the bundles of corticifugal fibers separate in their course through the pons, it has not been possible to differentiate well the location of the responses of the limbs. Lapping lies more and more laterally in these bundles. The last lapping was elicited on the dorsolateral surface of the bundles of corticifugal fibers immediately rostral to the formation of the pyramid (fig. 13). Stimulation of the fifth motor nucleus itself yields only a tonic closure of the jaws.

Since the corticifugal pathway for mastication, stimulation of which usually elicits lapping in the cat, may be followed back from the cortex in the internal capsule and basis pedunculi to the extreme caudal end of the pons, and since stimulation of the substantia nigra after degeneration of the corticifugal elements yields no trace of any masticatory reactions, it is no longer possible to regard the substantia nigra as an intercalary center in this pathway.

Incidental observations of other motor functions in the cat indicate the importance of the buried cortex on the lateral and dorsal banks of the presylvian sulcus as a motor area and demonstrate a ventromedial-dorsolateral distribution of the pathways concerned in motor responses of the head, anterior extremity and posterior extremity, in the internal capsule and rostral part of the basis pedunculi.

COLLOID TUMORS OF THE THIRD VENTRICLE

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Colloid cysts of the third ventricle, represented by twenty-eight reports in the literature, are among the less frequently encountered tumors of this region. Their position in the ventricular system, which permits them at times to block either or both foramina of Monro or the aqueduct of Sylvius, and their relation to the closely adjacent hypothalamic nuclei often give rise to a fulminating clinical picture. Sudden death is a frequent occurrence in tumors of this kind. Those cysts which interfere only intermittently with the circulation of the ventricular fluid and those which elicit but few symptoms referable to the vegetative centers offer difficult diagnostic problems.

Recently we had the opportunity to study two patients with this type of tumor whose cases presented some difficulties in diagnosis. It is our purpose to describe these cases in detail and to discuss the diagnostic and anatomic problems presented by colloid tumors of the third ventricle as a whole. In the accompanying table are summarized the important features of similar cases that have been reported.

REPORT OF CASES

CASE 1.—*Recurrent attacks of headache and weakness of lower extremities, of four years' duration, recently followed by periods of unconsciousness and signs of increased intracranial pressure. Ventriculogram. Two-stage removal of colloid cyst of third ventricle. Recovery.*

History.—W. L., a white man, aged 26, "night man" in a garage, was referred to the New Haven Hospital on Oct. 21, 1931, by Dr. W. H. Treat of Derby, Conn., with the chief complaint of recurrent attacks of headache and weakness of the lower extremities for four years, recently followed by loss of consciousness. The patient first began to have sudden severe attacks of bifrontal headache, associated with weakness of the lower extremities. The headaches lasted from a few minutes to an hour and were followed by slight drowsiness for several hours. The weakness was sufficient to require him to sit down, but he did not fall. At first the attacks occurred at intervals of about six months, but recently they had become more frequent and more severe and had been accompanied by projectile vomiting. There had been no aura preceding the attacks and no sensory or motor symptoms following them. During the six months before admission to the hospital, the attacks had occurred at monthly intervals and had been associated

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Chronological Tabulation of Colloid Cysts of the Third Ventricle

Case	Source	Sex	Age, Yrs.	Clinical Course	Description of Tumor
1.	Wallmann, H.: Virchows Arch. f. path. Anat. **14**: 385, 1858	M	52	Generalized muscular weakness, particularly of lower extremities; mental dulness; loss of sphincteric control; death with symptoms of "cerebral paralysis"	Cyst arose from choroid plexus medius and was covered by arachnoid (?)
2.	Stedmann, J.: Boston M. & S. J. **109**: 135, 1883	M	28	Headache; found dead in bed.....	Colloid cyst on velum interpositum, presumably of choroid plexus
3.	Batten, F. E., and Collier, J. S.: Brain **22**: 473, 1899 (case 29)	F	42	Giddiness and headaches for 14 days; vomiting and loss of consciousness; loss of sphincteric control; right pupil larger than left	Cyst of choroid plexus lying below anterior portion of fornix and blocking foramen of Monro
4.	Mott, F. W., and Barratt, J. O. W.: Arch. Neurol. **1**: 417, 1899 (case 1)	M	34	Duration 6 mos.; frontal headache; recurrent attacks of drowsy stupor, sometimes deepening to coma, preceded by restlessness and sometimes by convulsions; dementia paralytica; loss of memory; slowness of thought, speech and movement; ataxia; ptosis of left eye; double vision; sluggish pupils to light and in accommodation; loss of facial expression; slow pulse; double optic neuritis; vomiting; difficulty in swallowing toward end; deep coma	Cyst wall attached at one point to choroid plexus and probably arose from epithelial covering of latter
5.	Franceschi, F.: Riv. di pat. nerv. **9**: 457, 1904	F	26	Duration about 1 yr.; onset with drowsiness; patient weary, forgetful in her work; mental torpor; cessation of menses; brief period of vertigo, headache and occasional vomiting; failing vision; bilateral optic atrophy; panniculus adiposus; hypersomnia; memory poor; sluggish pupillary reflex on right and practically absent on left; positive Babinski reflex on right	Tumor size of large nut in third ventricle; author thinks it came from pia; no microscopic description
6.	Kauffmann, O. J.: Birmingham M. Rev. **57**: 231, 1905	M	40	Onset sudden; comatose, breathing deeply and stertorously; vomited earlier same day; left side of face flatter than right; slight weakness of left eyelids; urinary and rectal incontinence; mental confusion; frequent yawning; complete recovery; sudden loss of consciousness 21 days later; vomited once; coma deepened; face flushed; temperature, 104.6 F.	Cyst, size and shape of small hen's egg, apparently originating in choroid plexus of third ventricle, encapsulated in velum interpositum; author regarded growth as degenerated sarcoma, termed "myxosarcoma"
7.	Wilson, S. A. K.: Brain **29**: 524, 1906 (case 2)	M	42	Sudden onset with severe headache and vomiting 3 mos. before; loss of consciousness then and repeatedly subsequently; aura of giddiness for about 5 min.; urinary incontinence; repeated appearance of ectopia pupillae	Spherical tumor, size of large marble, apparently growing from ependyma and encapsulated in thin membrane
8.	Backer-Grøndahl, N.: Norsk mag. f. lægevidensk. **69**: 1058 1908	M	23	Could not stand hard work; some headache for years and trouble with vision; on day following intoxication had headache and vomiting; well next day but vomited during night and then died suddenly	Cyst suspended from roof of third ventricle extending from tela choroidea and attached to that of lateral ventricles
9.	Sjövall, E.: Beitr. z. path. Anat. u. z. allg. Path. **47**: 248, 1909	M	53	Headaches with striking remissions; vomiting only in last days	Cyst in posterior part of ventricle and attached to roof and plexus; lined by ciliated epithelial cells; author believes they are ependymal cells derived from paraphysis
10.	Weisenburg, T. H.: Brain **33**: 236, 1911 (case 3)	M	27	Frontal headaches, nausea and vomiting; progressive loss of vision; vision best when head was tilted backward; sight disappeared when head was bent forward or to side; death occurred suddenly	Colloid cyst, size of small marble, attached to choroid plexus in upper anterior portion of third ventricle posterior to middle commissure

11.	Hanson-Breide, H.: Hygiea, Med. & Farm. Monad. **73**: 1270, 1911 (Same case described by Sundberg, C.: Acta med. Scandinav. **59**: 423, 1923)	F	32	Headaches for about 1 yr., gradually increasing in severity, and usually in morning; these disappeared on lying down; severe attack with vomiting, loss of consciousness and rolling of eyes; six months later another severe attack followed by another on same day; next morning vomited again, became unconscious and died 15 min. later	Cystic structure, size of bean, in third ventricle; attached to choroid plexus
12.	Hall, A. J.: Lancet **1**: 89, 1913 (case 1)	M	Middle age	Found lying unconscious.....	Globular cystic tumor attached to anterior part of choroid plexus on right; fibrous pedicle formed capsule of cyst
13.	Hall, A. J.: Lancet **1**: 89, 1913 (case 2)	F	18	Patient died suddenly after 2 days of headache which confined her to bed	Tumor similar to that of case 12; attached to anterior right side of choroid plexus and rested on velum interpositum
14.	Pollock, L. J.: J. A. M. A. **64**: 1903 (June 5) 1915	F	48	Loss of memory for 6 mos.; gradual onset of stupor, apathy and somnolence; careless in appearance, untidy, filthy; loss of 20 lbs. in weight; slightly positive Romberg sign; shuffling gait; spasticity suggestive of extrapyramidal involvement; generalized weakness	Encysted ependymal growth attached to inferior surface of fornix and velum interpositum, occluding both formaina of Monro; capsule of connective tissue; author believed tumor to have originated from a glioma
15.	Beutler, A.: Berl. klin. Wchnschr. **57**: 667, 1920; Virchows Arch. f. path. Anat. **232**: 358, 1921	M	41	Headache; choked disks; vomiting; history of periodic seizures of headache and vomiting for years	Spherical cyst attached to choroid plexus and lined by ciliated epithelium; author called it ependymal cyst
16.	Bittorf: Berl. klin. Wchnschr. **57**: 667, 1920	?	?	Pupillary dilatation, tonic spasms, respiratory cessation and complete somnolence; fluttering pulse appeared when patient was placed on left side; these disappeared when patient was again placed on his back	Ependymal cyst of third ventricle, size of small apple, adherent to right thalamus
17.	Klarfeld: Berl. klin. Wchnschr. **57**: 667, 1920	?	?	Clinical diagnosis of brain tumor; no details.....	Ependymal cyst in third ventricle, extending through greatly dilated foramen of Monro into left lateral ventricle; lined by ciliated epithelium
18.	Buzzard, E. F., and Greenfield, J. G.: Pathology of the Nervous System, London, Constable & Co., Ltd., 1921	?	?	No clinical history or anatomic description.....	Illustration described as "colloid tumor of third ventricle, possibly derived from the pituitary"
19.	Risien-Russell, J. S.: Proc. Roy. Soc. Med. **14**: 50, 1921	M	36	Headache for 16 mos., worse when patient was tired; diplopia for 2 weeks; bilateral papilledema; diplopia on looking to extreme left; paralysis of left external rectus; left facial paresis; coarse tremor of outstretched hand; several fits ("queer in his manner") 1 week previously	Ball-like tumor composed of colloid with fibrous tissue capsule; no diagnosis made
20.	Schükry, I.: Ztschr. f. d. ges. Neurol. u. Psychiat. **86**: 488, 1923 (case 1)	M	46	Patient was an epileptic; mental dulness; difficulty in walking with left foot	Cyst, size of walnut, beneath fornix; wall of dense connective tissue lined by cuboid cells
21.	Wahlgren, F.: Acta path. et microbiol. Scandinav. **1**: 276, 1924	M	32	Recurrent frontal and temporal headaches for 2 yrs.; roentgenogram revealed marked thickening in region of left frontal sinus; unsuccessful attempts to drill into frontal sinus; poor pulse and respiration resulting in death	Cyst blocking foramina of Monro and attached by fine vessels to both choroid plexuses; lined by ciliated cells having dark brown granules on periphery; author inclined to believe that cyst is malformed paraphysis

Chronological Tabulation of Colloid Cysts of the Third Ventricle—Continued

Case	Source	Sex	Age, Yrs.	Clinical Course	Description of Tumor
22.	Guillain, G.; Bertrand, I., and Périsson: Rev. neurol. **32**: 467, 1925	M	29	Recurrent headaches, vomiting and failing vision for 1 yr.; sudden attack of deep somnolence lasting 36 hrs.; catatonia and polyuria during attack; repeated attacks subsequently with catatonia and increased reflexes; choked disks	Cyst wall continuous with choroid plexus and with roof of ventricle; lined by cuboidal epithelium
23.	Drennan, A. M.: Brit. M. J. **2**: 47, 1929 (case 1)	F	19	Headaches and faintness at intervals for about 2 yrs.; loss of consciousness and death following such attack	Cyst attached to choroid plexus at anterior end of third ventricle
24.	Drennan, A. M.: Brit. M. J. **2**: 47, 1929 (case 2)	M	23	No previous history of any kind; patient found groaning and barely conscious one morning; vomiting and involuntary bowel movement; died ¼ hour later	Cyst attached to adjacent part of choroid plexus of third ventricle; author believes cyst contents formed by activity of lining cells and partly by serous transudation; believes their origin to be from choroid plexus
25.	Fulton, J. F., and Bailey, P.: J. Nerv. & Ment. Dis. **69**: 18, 1929 (case 3)	M	38	Vertigo for 20 yrs.; sudden periodic attacks of frontooccipital headache for 3 yrs., twice associated with projectile vomiting and often relieved by tilting head backward; occasional slight visual disturbance; memory poor with transient attacks of retrograde amnesia; hypersomnia; left lower facial paresis; contraction of left temporal visual field; slight weakness on left side; ventriculography showed dilated lateral ventricles and no filling of third ventricle; suboccipital exploration; lumbar puncture 2 mos. later; temperature, 109 F.; death	Cyst attached to roof of third ventricle and lined by cuboidal epithelium resembling that of choroid plexus; diagnosed cyst of choroid plexus
26.	Kaufmann, E.: Pathology for Students and Practitioners, translated by Stanley P. Reimann, Philadelphia, P. Blackiston's Son & Co., 1929, vol. 3, p. 1841	M	43	Symptoms of varying degrees of increased intracranial pressure; no further details	Cyst inserting in tela choroidea behind anterior commissure
27.	Hassin, G. B., and Anderson, J. B.: U. S. Vet. Bur. M. Bull. **6**: 56, 1930	M	35	Numbness and itching in head and left side of face; paresthesias lasting 5 to 15 min. and sometimes accompanied by vomiting and muscular weakness, often causing patient to fall; headaches; lacrimation (left eye); memory defect; attacks of severe headache accompanied by loss of consciousness and muscle weakness; tendon reflexes hyperactive; mild paresis of face on lower right side; bilateral papilledema; roentgenographic and stereoscopic examinations of skull negative; right subtemporal decompression; tumor not found; high voltage roentgenotherapy without avail	Tumor enclosed within and adherent to septum pellucidum and resting on middle commissure; choroid plexus and tela formed outer covering of tumor's capsule; no inner epithelial lining; diagnosed cystic tumor of septum pellucidum
28.	Byrom, F. B., and Russell, D. S.: Lancet **2**: 278, 1932	F	49	Intense hunger and extreme lassitude for 20 yrs.; vague headaches; extreme drowsiness lasting several days; sugar and acetone in urine; treated with insulin; recurring attacks of headache and tinnitus; emotional instability; subnormal temperature; headache followed by rapid loss of consciousness; lack of dehydration; respiratory failure; vigorous treatment with insulin of no avail	Ependymal cyst of roof of third ventricle projecting from lower surface of fornix into third and left lateral ventricles; attached to tela choroidea and choroid plexus of left lateral ventricle; lined by ciliated epithelium containing blepharoplasts; authors conclude that it is "an ependymal and not a choroid plexus cyst"

with flushing and sweating, especially in the upper portion of the body and face. In the last six weeks the patient had experienced the attacks at weekly intervals, and the last two seizures had been followed by unconsciousness for twelve and eighteen hours, respectively. Recently, he had been constantly disturbed by bad dreams while asleep. Blurring of vision had been noted during the past two weeks. He had continued his work until one week before admission and had been entirely free from headaches and narcolepsy in the intervals between attacks. There had been no convulsive seizures at any time, and there was no polyuria,

Fig. 1 (case 1).—Ventriculogram showing internal hydrocephalus and absence of air in the third ventricle. Note the upward bulge of the floor of the lateral ventricles directly above the third ventricle.

polydypsia or increase in weight. At the time of admission to the hospital he was unconscious, eighteen hours after the onset of the last attack. He was right-handed.

Examination.—Following intravenous injection of hypertonic dextrose solution, the examination gave entirely negative results except for papilledema of 4 diopters of the right optic disk; visual acuity was 20/30; a scar of the left cornea precluded ophthalmoscopic examination; the cremasteric reflexes were absent bilaterally.

The impression was that the condition was tumor of the third ventricle.

Ventriculography (fig. 1).—On October 22, ventriculography revealed internal hydrocephalus of both lateral ventricles. The third ventricle did not fill with air,

and there was an upward bulge of the floor of the lateral ventricles directly above the third ventricle.

First Operation.—On October 23, with the patient under avertin and procaine hydrochloride anesthesia, a right frontal osteoplastic bone flap was elevated by Dr. Samuel C. Harvey; the ventricle was tapped and the dura opened widely. The cortex was transected with the electrosurgical unit anterior to the precentral gyrus and the ventricle opened. A gray-white tumor was seen bulging into the foramen of Monro. A large subtemporal decompression was left and the bone flap replaced.

Postoperative Course.—The patient recovered rapidly following the operation, with occasional elevations of temperature to 101 F. during the first week only. The fluid intake varied between 2,750 and 4,300 cc.; the output was from 1,600 to 3,270 cc. On November 7, the patient had a sudden attack of weakness, without headache, and the temperature rose to 101.2 F. The subtemporal decompression was bulging and moderately tense during this period.

Second Operation.—On November 19, with the patient under avertin and procaine hydrochloride anesthesia, the bone flap was reelevated by Dr. Harvey, the dura and the cortex were reopened, and a tumor about 2 cm. in diameter was seen in the third ventricle. The foramen of Monro was enlarged posteriorly, and the capsule of the tumor was opened. It contained thick gelatinous fluid which was removed with a sucker. The tumor was attached to the choroid plexus of the third ventricle at the right foramen of Monro. The attachment of the collapsed cyst to the choroid plexus was clipped and cut, completing the removal. The ventricle was filled with physiologic solution of sodium chloride, the dura closed and the bone flap replaced.

Postoperative Course.—Again there was a slight elevation of temperature to 101 F. for the first week, but no loss of consciousness, disturbance of water balance or marked fluctuation in blood pressure. The patient was allowed up on the eighth postoperative day and was discharged from the hospital on December 2. The subtemporal decompression was still bulging slightly but was not tense. There remained 2 diopters of papilledema and slight secondary atrophy; visual acuity was 20/25.

Subsequent Course.—On December 15, the patient reported no recurrence of symptoms and wished to return to work. The papilledema had completely subsided, and the decompression was soft and flat. On March 22, 1932, the patient reported that he had returned to work in January and had been symptom-free. He had also enjoyed complete freedom from the bad dreams which he previously experienced. On May 17, he reported that he had been working regularly and had been completely free from all the previous symptoms. In August, 1932, the patient fell from a window and was killed, but up to the time of death there had been no evidence of a return of the previous symptoms. Permission for a postmortem examination was not obtained.

Description of Specimen Removed at Operation.—The wall of the cyst was composed of highly vascularized connective tissue lined internally by epithelium, 3 or 4 cells in thickness. Papillae or rugae of connective tissue, covered by this epithelium, projected into the cavity of the cyst (fig. 2). To one part of the wall was attached a piece of choroid plexus which was covered by the usual flat cuboidal epithelium. The cells lining the inner surface of the cyst were flattened from side to side and some had dark hyperchromatic nuclei, while others had pale

vesicular nuclei. The cells on the free surface were of columnar shape and contained cilia (fig. 3). In the cytoplasm of these cells at the base of the cilia were occasional small, dark blepharoplasts.

The thick gelatinous fluid forming the contents of the cyst was of a milky color and contained a few small and large mononuclear cells. With eosin this material stained a homogeneous pale pink; stains for mucin were unsuccessful.

Comment.—The sudden severe headache, weakness and, later, unconsciousness were undoubtedly due to displacement of the pedunculated tumor of the third ventricle posteriorly, blocking the outlet of the

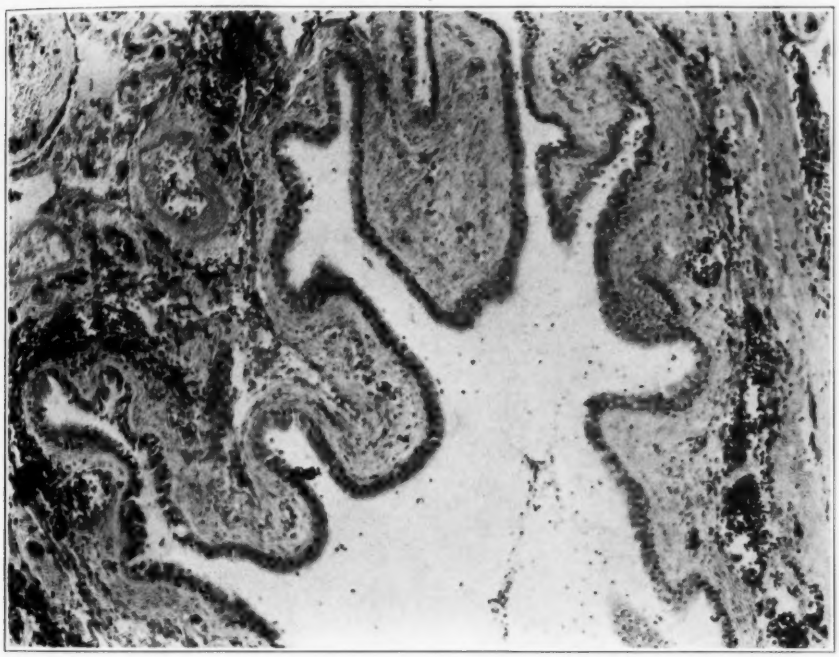

Fig. 2 (case 1).—Photomicrograph of the wall of the cyst. Note the papillae of connective tissue projecting into the lumen of the cyst and covered by epithelium. Heidenhain's hematoxylin; × 100.

third ventricle. The frequent symptoms of a tumor in this region, diabetes insipidus and narcolepsy, were absent, but the attacks of unconsciousness associated with flushing and sweating correspond closely with the symptoms in Penfield's case.[1] Blurring of vision appeared only shortly before the patient's admission to the hospital, indicating that the marked obstruction of the third ventricle was of relatively recent occurrence.

1. Penfield, W.: Diencephalic Autonomic Epilepsy, Arch. Neurol. & Psychiat. **22**:358 (Aug.) 1929.

The absence of hyperpyrexia, sympathetic overaction and unconsciousness immediately following operation may be explained by the fact that the tumor was not adherent to the walls or floor of the third ventricle, and these structures were not disturbed at operation. If the hydrocephalus is of a considerable degree, as it was in this case, it would appear justifiable to transect the thinned-out cortex and open the lateral ventricle at the first stage of the operation. If this is not the case, it would certainly be better judgment simply to raise a large bone flap and leave an ample decompression to allow sufficient dilatation of

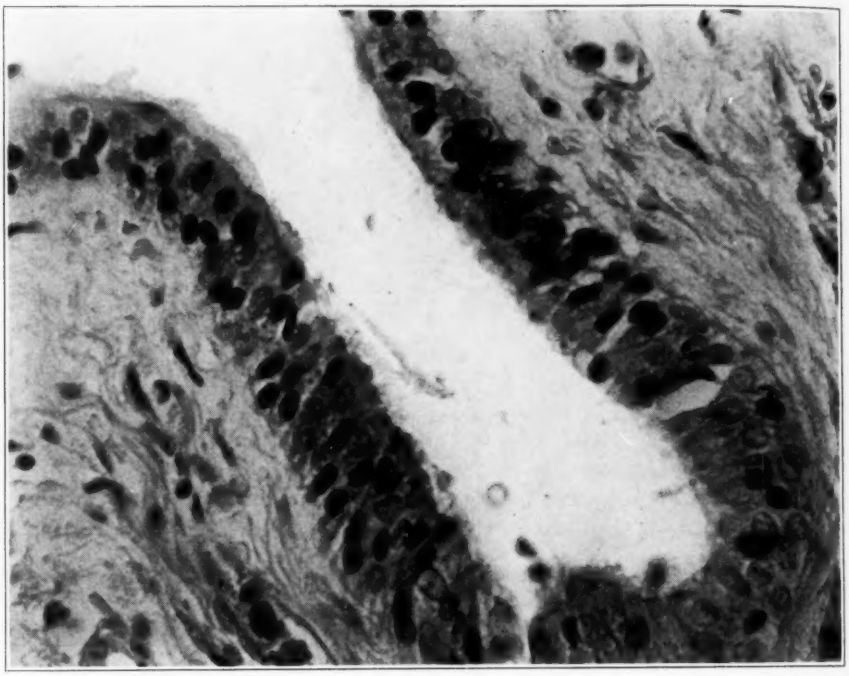

Fig. 3 (case 1).—Photomicrograph of epithelium lining inner surface of the wall of the cyst. Note the cilia. Heidenhain's hematoxylin; × 600.

the ventricle on that side. The fact that the contents of the tumor could be removed by suction minimized the trauma to the adjacent structures at operation.

CASE 2.—*Blurred vision, headache, vomiting and unsteadiness. Signs of increased intracranial pressure and slight ataxia. Emergency ventricular puncture. Ventriculogram. First stage operation for tumor of third ventricle. Death two days later. Postmortem examination revealed colloid cyst in third ventricle.*

History.—P. R., a white man, a clerk, aged 22, was referred to the New Haven Hospital on Feb. 15, 1932, by Dr. Z. A. Bonoff of New Haven, Conn., with the chief complaint of headache, vomiting and unsteadiness of gait. About eight

months before, he first noticed slight blurring of vision, which had increased rapidly during the past two months. At the same time he began to have brief dizzy spells which continued. Two months before, he began to complain of frontal headaches, which later radiated posteriorly and finally became localized in the suboccipital region. Five days before, he experienced sudden severe occipital headache and vomited. The severe headache and vomiting had continued intermittently since that time, and the patient had been quite drowsy, with diplopia on one occasion. On the day of admission to the hospital he complained of unsteadiness of gait and weakness of the lower extremities. He had been confined to bed at home for the past six days. There was no history of convulsive seizures, diabetes insipidus, narcolepsy, unconsciousness, flushing or sweating. The patient was right-handed.

Examination.—The patient was moderately drowsy and complained of headache. There was bilateral papilledema of from 5 to 6 diopters, with considerable peripapillary edema. The pupils were moderately dilated and reacted sluggishly to light. The left pupil was slightly larger than the right, and there was slight limitation of upward gaze. There were no ocular nerve palsies or nystagmus and no gross defect of the visual fields. There were slight right facial paresis of the supranuclear type and suggestive weakness of the right upper extremity. The deep reflexes were slightly more active on the right side, and the abdominal reflexes were more easily fatigued on the right. The gait was unsteady, with a tendency to veer to the right. The head was held slightly to the right with the chin pointed toward the left shoulder.

The impression was that the condition was tumor of the fourth ventricle.

Ventricular Puncture.—The patient vomited several times on the day after admission to the hospital and became extremely drowsy. Ventricular puncture on February 16 revealed internal hydrocephalus with a pressure of 450 mm. of water. The condition immediately improved, and he became once more responsive.

Ventriculography.—On February 17, air injected through a single needle in the right lateral ventricle revealed hydrocephalus of this ventricle but no air in the left or third ventricles (fig. 4). The air failed to cross from the right to the left lateral ventricle when the head was turned with the left side up (fig. 5). Subsequent puncture of the left lateral ventricle revealed only slight dilatation of that cavity.

The impression at this time was that the condition was tumor of the third ventricle blocking the right foramen of Monro.

Operation.—On February 17, with the patient under procaine hydrochloride anesthesia, a right frontal osteoplastic bone flap was elevated, the ventricle tapped and the dura opened. The cortex was transected with the electrosurgical unit, well anterior to the precentral gyrus, and the ventricle opened. The cortex was not as thin as had been expected from ventriculography. The septum pellucidum was bulging into the ventricle in such a manner that the foramen of Monro could not be visualized. The septum was punctured and was found to contain what appeared to be a fifth ventricle, filled with clear fluid. With the intention of exploring the third ventricle at a second operation, a large subtemporal decompression was left and the wound was closed. The patient's pulse rate gradually rose during the operation, from 64 to a maximum of 160 per minute; it was 120 when the closure was completed; the blood pressure remained unchanged.

Postoperative Course.—The patient remained unconscious for two days following operation. During this period he had several crises of flushing, sweating, elevated blood pressure, slow pulse with extrasystoles, and rapid respirations followed by low blood pressure, rapid pulse, cyanosis and slow, irregular respira-

Fig. 4 (case 2).—Ventriculogram showing hydrocephalus of the right lateral ventricle.

Fig. 5 (case 2).—Ventriculogram showing the failure of air to cross from the right to the left ventricle when the head is held left side up.

tions. During each of these crises, ventricular puncture revealed increased pressure and relieved the attack. The temperature varied between 101 and 103 F. On the second day after operation, only a small amount of fluid was obtained by ventricular puncture during one of these attacks; the respirations ceased, and the patient died.

Necropsy.—Postmortem examination was limited to the intracranial contents. At the site of the operative wound over the right frontal lobe an extradural hemorrhage was present, but the dural vessels of the region were intact. Small wounds were present in both the left and the right occipital lobe at the sites of the ventricular punctures. The cerebral convolutions were flattened, and a large cerebellar pressure cone was wedged into the foramen magnum. Both lateral ventricles were dilated, the right to a less degree than the left.

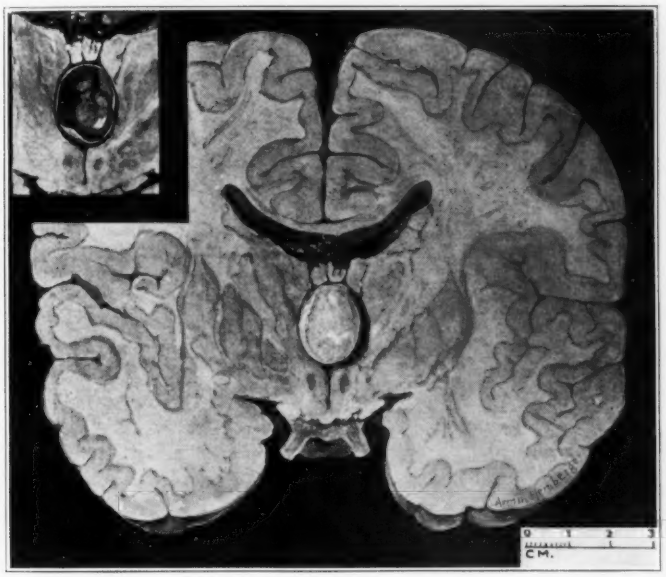

Fig. 6 (case 2).—Drawing of the gelatinous tumor in the upper part of the third ventricle lying beneath the corpus fornicis. Note the thin wall of the cyst after the content is evacuated.

A gelatinous oval tumor mass was present in, and completely filled, the upper part of the greatly dilated third ventricle (fig. 6). The tumor was soft and translucent when first exposed, but soon acquired a firm, rubbery consistence when placed in a diluted solution of formaldehyde, U.S.P. (1:10). It was completely encapsulated in a thin, translucent capsule which was adherent to, and continuous with, the choroid plexuses of the lateral ventricles at the foramina of Monro. The solidified gelatinous mass could be evacuated from its capsule, to which it was nowhere adherent. When this was done it maintained its shape and had an appearance which might best be described as that of a Malaga grape. The tumor lay beneath the corpus fornicis without being attached to this structure, and compressed the anterior and lateral thalamic nuclei as well as the upper part of the periventricular gray matter. The lower part of the third ventricle was not even

dilated and, of course, the tuberal nuclei were not encroached on. The capsule of the tumor could be peeled readily from the walls of the ventricle, its only points of attachment, as already mentioned, being the choroid plexuses at the foramina of Monro. The ventricular wall appeared to be lined by a smooth and glistening membrane after the capsule of the tumor was removed.

The choroid plexuses in the lateral and fourth ventricles were not abnormal in any way.

On microscopic examination the wall of the cyst was seen to be composed of connective tissue lined internally by a single layer of flattened, nonciliated, cuboidal cells which contained no blepharoplasts (fig. 7). The colloid-like material within the cyst had been dehydrated preparatory to embedding and became quite brittle. It fragmented into innumerable pieces when sectioned with the microtome. Stained

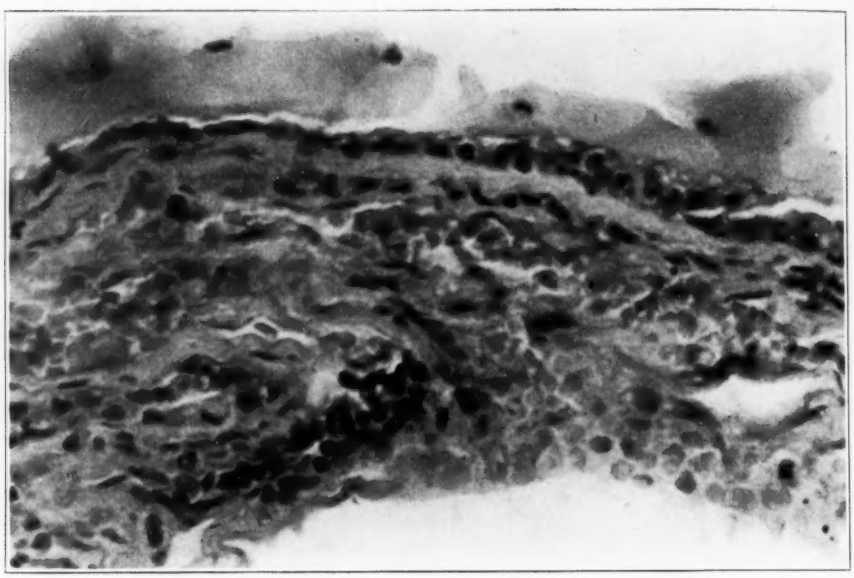

Fig. 7 (case 2).—Photomicrograph of the wall of the cyst showing the connective tissue lined by low cuboidal epithelium. Note the homogeneous colloid-like material adjacent to the latter and containing large mononuclear cells. Hematoxylin-eosin; × 550.

with hematoxylin-eosin, this material had a homogeneous dark red color and contained a few large mononuclear cells.

In Nissl preparations the ependymal lining of the third ventricle was everywhere intact. Interesting lesions associated with the increased intracranial pressure were present in many parts of the cerebral cortex, and consisted of widespread foci of necrobiosis. Sometimes one and sometimes another of the cerebral cortical laminae was destroyed, and sometimes all six laminae were destroyed in the same region. In many places in the cortex the blood vessels were greatly congested and often ruptured with the production of focal hemorrhages. Nerve cells on the periphery of the necrobiotic foci showed, besides the more usual ischemic changes, shrinkage, hyperchromia and pericellular incrustation.

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Comment.—This case presented a difficult diagnostic problem, the symptoms and signs being more suggestive of a tumor of the fourth ventricle. However, the patient's age and the paucity of cerebellar manifestations made ventriculography advisable. The necessity of an emergency ventricular puncture and the failure of air to cross from one lateral ventricle to the other indicated an advanced degree of obstruction in the third ventricle and probably a poor prognosis. In view of the thickness of the cerebral cortex found at operation, it would undoubtedly have been better judgment to postpone cortical transection to a second stage operation, limiting the first operative procedure to a bone flap with a large decompression. The subsequent dilatation of the ipsilateral ventricle would have minimized the cortical transection at the second stage. The presence of a dilated fifth ventricle was probably a minor factor in the production of the symptomatology, since evacuation of this structure afforded no relief. The stormy postoperative course and ultimate fatal outcome were undoubtedly related to the size of the tumor and the edema around it, as well as to its possible displacement at operation, and the sympathetic crises were probably produced by the same causes.

The presence of extrasystoles during these attacks is interesting in the light of the recent observations of Beattie, Brow and Long,[2] who found that the extrasystoles induced experimentally by chloroform anesthesia could be abolished by section of the hypothalamus. They found, further, that stimulation of the posterior part of the lateral wall of the third ventricle produced an extrasystolic arrhythmia in an animal under chloroform anesthesia. From these and other observations they concluded that the hypothalamic nuclei and their fiber tracts control this sympathetic phenomenon.

COMMENT

In many ways the symptomatology presented by colloid cysts of the third ventricle is similar to that presented by any tumor in this region. Weisenburg called particular attention to the following major symptoms of tumors in this ventricle: (1) paralysis of associated ocular movements, especially of upward convergence but also less commonly of lateral and downward convergence; (2) cerebellar ataxia, particularly prominent in the lower extremities; (3) signs of involvement of the pyramidal tracts, and (4) mental dulness and drowsiness. The latter symptoms were attributed by him to "compression of the cortex against the skull resulting from internal hydrocephalus." Högner [3] attempted

2. Beattie, J.; Brow, G. R., and Long, C. N. H.: Physiological and Anatomical Evidence for the Existence of Nerve Tracts Connecting the Hypothalamus with Spinal Sympathetic Centres, Proc. Roy. Soc., London, s.B, **106**:253, 1930.

3. Högner, P.: Die klinischen Erscheinungen bei Erkrankungen des 3. Gehirnventrikels und seiner Wandungen, Deutsche Ztschr. f. Nervenh. **97**:238, 1927.

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to divide the symptoms produced by lesions in this region into two main groups; namely, general symptoms of increased intracranial pressure, and local symptoms referable to the position of the lesion itself. Among the general symptoms he listed headache, vertigo, malaise, vomiting, psychic disturbances, apathy, coma, pulse deficits, bradycardia, papilledema, optic "neuritis," disturbances of speech, smell and hearing, obstipation, ocular palsies, ataxia and tonoclonic convulsions. The local symptoms were grouped as follows: (1) motor and sensory changes, such as epileptiform attacks, paresthesia, paresis, spasticity, masked countenance, athetosis and chorea; (2) vegetative disturbances, as vasomotor changes, excessive perspiration and salivation, exophthalmos and dilatation of the pupils; (3) disturbances of bladder and intestinal innervation; (4) disturbances in heat regulation; (5) disturbances in the metabolism of carbohydrates, salt and water; (6) disturbances in sleep, and (7) sudden death. In an extensive review of this whole question, Fulton and Bailey emphasized that there is no syndrome of the third ventricle per se, but that tumors of this region cause the characteristic symptoms just enumerated by pressure on the adjacent nuclei and fiber tracts.

A proper evaluation of the symptomatology of the reported cases of colloid cysts of the third ventricle must take into account the fact that in but few instances are the histories complete in all details. Reference to the accompanying table will show that negative findings are entirely unmentioned and that several of the histories present few or no positive findings. Furthermore, it is not permissible to assume that the stated symptoms were the only ones presented by the patients. With these facts in mind it is surprising with what frequency, nevertheless, certain of the findings recur in a review of these cases.

General symptoms of increased intracranial pressure as manifested by headache were present in eighteen instances (cases 2, 3, 4, 5, 7, 8, 9, 10, 11, 13, 15, 19, 21, 22, 23, 25, 27 and 28). Vomiting was present in fourteen (cases 3, 4, 5, 6, 7, 8, 9, 10, 11, 15, 22, 24, 25 and 27); vertigo in four (cases 3, 5, 7 and 28); loss of consciousness in nine (cases 3, 6, 7, 11, 12, 23, 24 and 27), and mental dulness, loss of memory or confusion in eight (cases 1, 4, 5, 6, 14, 20, 25 and 27). Diplopia was present in two (cases 4 and 19); ptosis of the eyelids in two (cases 4 and 6); failing vision, optic neuritis or atrophy, or papilledema in nine (cases 4, 5, 8, 10, 15, 19, 22, 25 and 27). Apathy, drowsiness, stupor, somnolence or coma was described as occurring in cases 5, 6, 14, 16, 22 and 28. Changes in the pulse were present in cases 4, 16 and 21; tonic spasms in case 16, and convulsions in case 4.

Localizing symptoms of a motor and sensory nature were present in eleven cases. These took the form of facial paresis in cases 6, 19, 25

and 27; masked countenance and dementia paralytica in case 4; extrapyramidal spasticity, shuffling gait and a positive Romberg sign in case 14; muscular weakness in cases 1, 8, 14, 20, 25, 27 and 28; increased reflexes in cases 22 and 27, and paresthesia in case 27. Vegetative disturbances, manifested by changes in the pupils, were found in cases 3, 4, 5, 7 and 16. Lacrimation was seen in case 27. Loss of sphincteric control was noted in cases 1, 3, 6, 7 and 24. Disturbances in metabolism producing polyuria were found in case 22; in case 5 the menses ceased with the onset of symptoms of tumor of the brain, and the patient became adipose. Hypersomnia was definitely present in cases 4, 5 and 25, and was possibly also present in patients described as apathetic, stuporous and comatose. Frequent yawning was noted in case 6. Sudden death occurred in several instances (cases 2, 8, 10, 11, 13, 23 and 24).

Two important features in the clinical picture presented by colloid cysts of the third ventricle need emphasis. One is the almost uniform sudden onset of symptoms, which is dramatic and is probably to be associated with the sudden blocking of either the foramina of Monro or the aqueduct of Sylvius by the movable cyst. The other is frequency of remissions in the symptomatology, which again is attributable to the movability of the cyst producing an unblocking of the passageways of the cerebrospinal fluid. Here it is important to note that improvement in symptomatology could be produced almost at will in cases 10, 11, 16 and 25 by tilting the head or by lying down. This, however, is sometimes true of other intracranial tumors, particularly those of the posterior fossa.

As knowledge of the functions of the various nuclear masses and fiber tracts around the third ventricle increases, it becomes easier to interpret the various symptoms produced by pressure on these structures from tumors in the cavity. It is not difficult to understand why there is a certain degree of variation in the histories just reviewed, for obviously tumors of different size and in different parts of the third ventricle will implicate different structures and therefore produce different symptoms. Högner, as well as Hassin and Anderson, called attention to the paucity of symptoms pointing to involvement of the third ventricle when the tumor lies in the upper part of this cavity and is attached only to the roof. By contrast, the symptomatology pointing to involvement of this region is much more striking when the tumor exerts pressure on the structures in the lateral walls and floor of the cavity.

With the exception of Fulton and Bailey's case and the case of Hassin and Anderson, ventriculography was not utilized in the localization of the tumor. In the former instance this procedure failed to show air in the third ventricle, but in spite of this a cerebellar operation was performed with negative results. In the latter instance, ventriculo-

graphy failed to disclose the growth. The tumor was distinctly localized, however, in each of our two cases by this method, and it would therefore appear justifiable in the future to subject any patient with symptoms suggestive of tumor in this region to this procedure.

From a pathogenic point of view the colloid cyst of the third ventricle presents few but none the less interesting possibilities. The majority of writers consider it to be a tumor of the choroid plexus of the third ventricle. This seems to be supported by the fact that the capsule is composed of connective tissue and is lined by cuboid cells similar to those covering the plexus. Also, its only point of attachment usually seems to be to the plexus of this ventricle, and usually to the velum interpositum. Occasionally, as in our case 2, the attachment is to the choroid plexus at one or both foramina of Monro, at the point called the glomus choroideum.

On the outer surface of the roof of the third ventricle is a fold of pia mater in the transverse fissure known as the tela choroidea. Franceschi apparently utilized this pial fold in explaining the origin of his tumor when he stated that it was derived from pia. However, as the tela choroidea gives rise to the vascular folds which are invaginated into the ventricle to form the choroid plexus proper, the question as to whether the tumor is of pial or choroidal origin is only of academic interest.

It is the opinion of a number of writers that these growths represent ependymal tumors. This is based on the fact that a certain number of them are lined internally by ciliated epithelium. That the ependyma may be the seat of origin of such tumors cannot at present be denied, but it is interesting to note that embryologically the cells covering the choroid plexus as well as the ependymal cells bear cilia.

In front of the velum transversum in the roof plate of the primitive forebrain is an outgrowth, the paraphysis, which arises on the dorsum of the brain in nearly all vertebrates. The paraphysis is non-nervous and is apparently an extraventricular choroid plexus with secretory functions. To this structure, which is quite rudimentary in man, both Sjövall and Wahlgren are inclined to attribute the origin of the colloid cysts which they have described. Sjövall stated that his tumor "was found upon the velum interpositum in the median line behind the fifth ventricle in the region of the anterior commissure." This is a happy description of the position of the paraphysis.

Hassin and Anderson believed their case to be a cystic tumor of the septum pellucidum. These writers failed to find an inner epithelial lining in their cyst, but the gross characteristics of the latter, such as color, consistence and attachment to the choroid plexus and tela, are similar to those in the other cases described. On the whole, these cysts

are not to be confused with the cystic dilatations of the cavum septi pellucidi described by Dandy.[4]

A few other opinions are entertained by different writers as to the nature of the cysts. Kaufmann regarded it "as being a degenerated sarcoma, probably most fittingly termed a myxo-sarcoma." Pollock believed his tumor to have originated from a glioma, and Buzzard and Greenfield described theirs as a "colloid tumor of the third ventricle, possibly derived from the pituitary."

SUMMARY

Two instances of colloid cyst of the third ventricle are presented with their detailed clinical histories and anatomic findings. Twenty-eight similar cases from the literature are tabulated, with a summary of the clinical features and essential pathologic findings in each. Attention is called to certain symptoms of diagnostic aid in the localization of the lesion, and emphasis is laid on the localizing value of ventriculography, which procedure was eminently successful in both original cases described. It is concluded that these cysts are usually derived from the choroid plexus of the third ventricle, but the possibility is conceded that in certain instances they may be derived from the ependyma lining this cavity. It also appears possible that rarely such a tumor may be derived from a persistent paraphysis.

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4. Dandy, W. E.: Congenital Cerebral Cysts of the Cavum Septi Pellucidi (Fifth Ventricle) and Cavum Vergae (Sixth Ventricle): Diagnosis and Treatment, Arch. Neurol. & Psychiat. **25**:44 (Jan.) 1931.

MYELITIC AND MYELOPATHIC LESIONS

IV. TRAUMATIC LESIONS OF THE SPINAL CORD; A CLINICO-PATHOLOGIC STUDY

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Trauma may produce a myelopathy by: (1) a direct injury to the cord proper by the traumatizing agent; (2) compression of the cord and its vessels (by a bullet, fractured or dislocated vertebrae, hemorrhages, etc.), with secondary circulatory interference, and (3) concussion of the cord.

REPORT OF CASES

CASE 1.—P. J. C., a man, aged 26, was admitted to the Broad Street Hospital on Dec. 16, 1925, immediately after having been shot in the right axilla. He was in shock from a hemorrhage in the axilla. Roentgen examination showed a fracture of the fourth rib. The point of exit of the bullet was below the angle of the left scapula. Twenty hours later, there appeared harsh breathing over the apexes of both lungs, with subcutaneous emphysema over the left side of the chest.

Neurologic Examination.—There were complete flaccid paraplegia, with absent knee and ankle jerks, no pathologic reflexes, rigidity of the abdominal muscles, absence of the abdominal reflexes on the left side, diminished cremasteric reflex on the right and partial priapism. Sensory examination revealed a zone of hyperalgesia between the sixth and eighth dorsal segments, below which there was complete loss of all forms of sensation.

A diagnosis of crush with incomplete severance of the cord at about the seventh and eighth dorsal segments was made. Exploratory laminectomy was advised and attempted on the next morning, but the patient died before the operation was completed, fifty-one hours after the injury.

Laboratory Data.—The urine contained albumin, hyaline and granular casts and a moderate number of pus cells. The Wassermann reaction of the blood was negative.

The clinical diagnosis was gunshot wound of the lung; laceration of the spinal cord.

Autopsy.—Gross Examination: A piece of the spinal cord, 12 cm. long, was received for examination. The meninges and vessels were hyperemic. The pia-arachnoid was adherent to the cord. At about the middle of the specimen the posterior surface of the cord disclosed a lesion (1 by 0.5 cm. long) in which the meninges were torn and the substance of the cord was soaked with blood.

Microscopic Examination: Sections above the lesion showed: The contour of the cord appeared well preserved. There were slight swelling of the myelin sheaths

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From the Neuropathologic Laboratory, Montefiore Hospital.

throughout the whole section, and diffuse recent hemorrhages in the anterior, lateral and posterior columns. The meninges were edematous and infiltrated with numerous lymphocytes and polymorphonuclear leukocytes, which were also present near the hemorrhagic foci. The anterior horn cells appeared normal.

Sections at the lesion showed: The structure of the cord could not be distinguished (fig. 1). The section consisted chiefly of red blood cells, with numerous engorged vessels; in some of these the adventitia was surrounded by a ring of red blood cells (fig. 2*A*), in others also by lymphocytes and polymorphonuclear leukocytes (fig. 2*B*), and in still others only by polymorphonuclear leukocytes. The meninges were edematous and thickened, with hemorrhages and engorged vessels

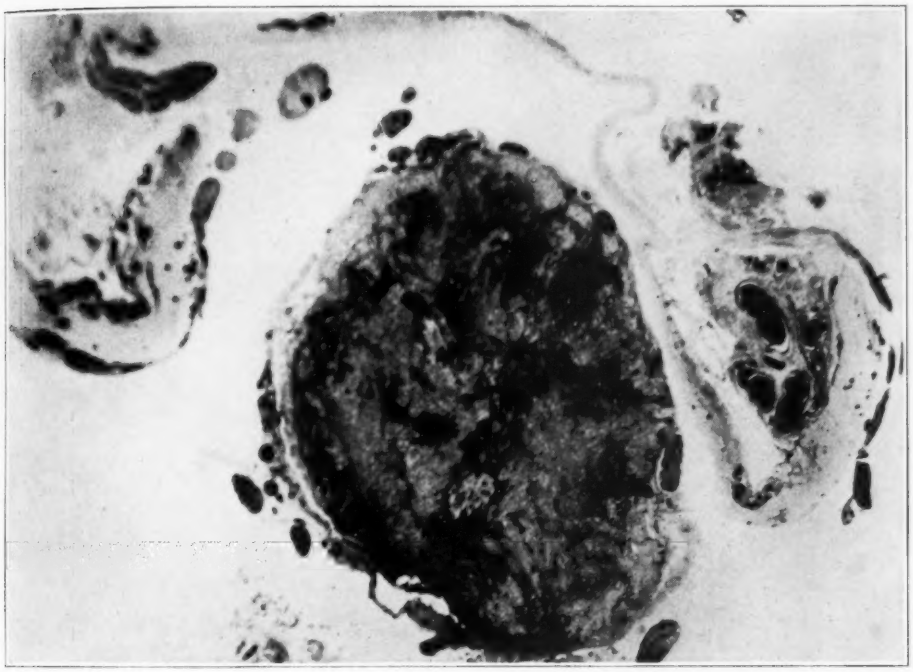

Fig. 1.—Transverse section of the cord at the level of the lesion. The structures of the cord cannot be distinguished; the entire cord is full of hemorrhages. Myelin sheath stain (Weil modification).

containing polymorphonuclear leukocytes and lymphocytes. Some of the anterior horn cells were in the stage of swelling, with complete absence of Nissl substance and eccentric nuclei, while others were completely destroyed (fig. 3). All myelin sheaths showed marked swelling and beginning disintegration. The axis cylinders within the hemorrhagic foci (fig. 4*A*) were completely disintegrated, while those near these areas showed only slight destruction (fig. 4*B*). In sections stained with Mallory's phosphotungstic acid the glia within the hemorrhagic areas could not be distinguished, but in the adjacent areas it was fairly normal; apparently the pathologic process was too recent to produce glial changes.

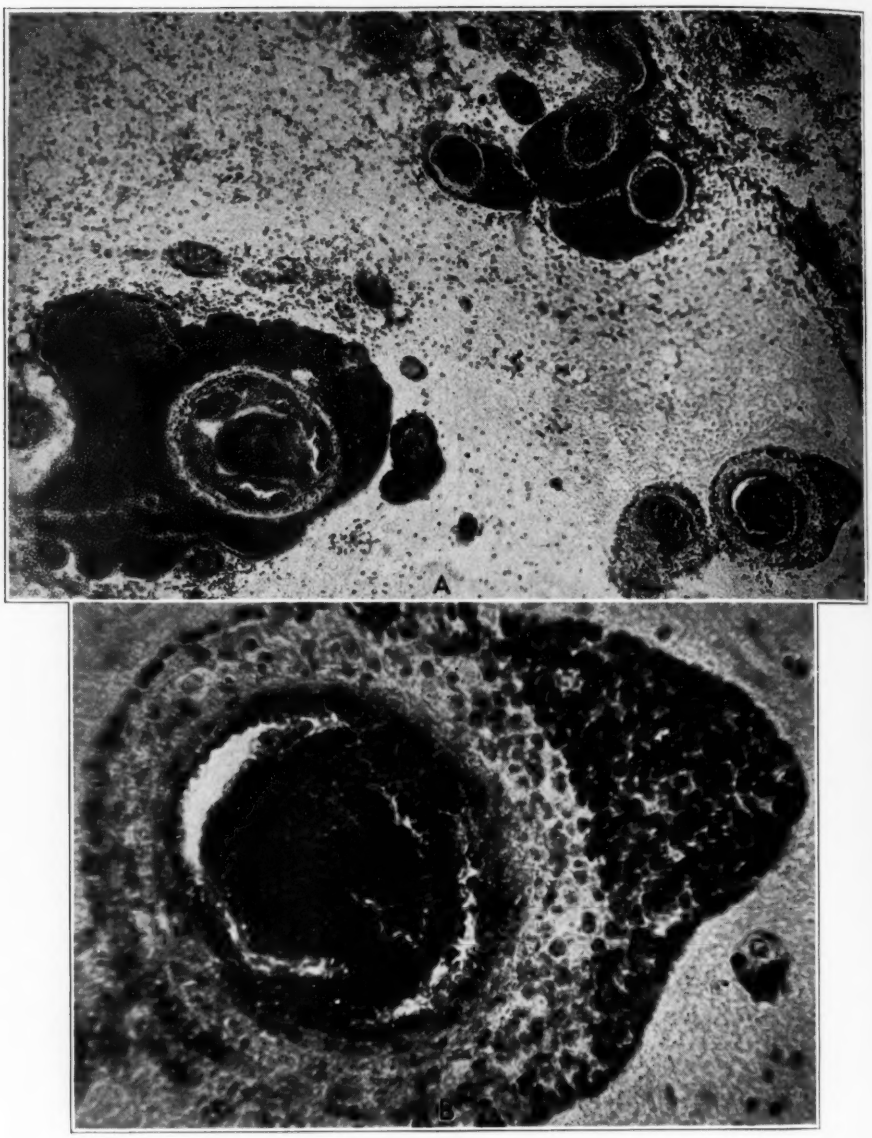

Fig. 2.—*A*, ring of red blood cells about the vessels of the cord. Notice the ring of white blood cells in the two vessels to the right. Hematoxylin-eosin stain; reduced from × 150. *B*, ring of lymphocytes, polymorphonuclear leukocytes and red blood cells about a blood vessel. Hematoxylin-eosin stain; reduced from × 400.

Sections below the lesions showed: There were extensive hemorrhages in the posterior columns and smaller foci scattered throughout the whole cord.

The microscopic diagnosis was myelopathy, traumatic.

Comment.—In addition to the trauma, the cord showed evidences of an inflammatory reaction (cellular infiltration). This was due to two factors: (1) an immediate defense reaction by a mobilization of inflammatory cells to the traumatized area, and (2) possibly a meningeal reaction to the injury.

CASE 2.—M. R., a woman, aged 55, was struck by a taxicab and hurled into an excavation, 20 feet deep, on July 20, 1926. She was taken to Lincoln Hospital where she remained for six weeks; a diagnosis was made of fracture of the vertebrae and

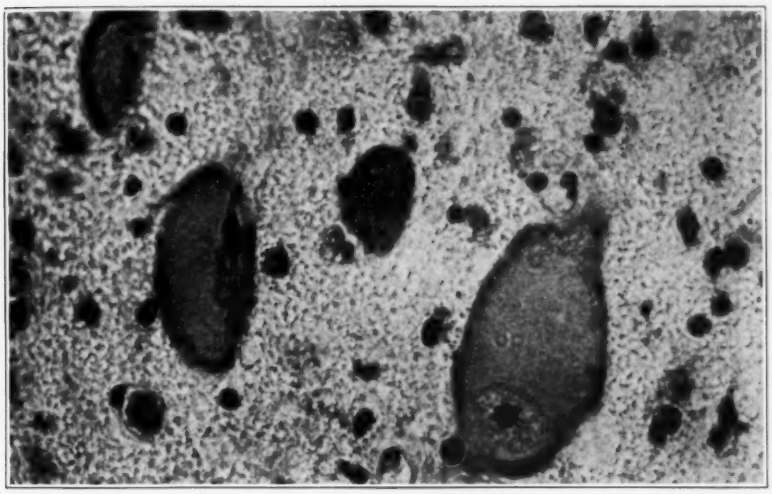

Fig. 3.—Anterior horn cells in a state of swelling, with complete loss of Nissl substance and eccentric nuclei. Cresyl violet stain; × 400.

transverse myelitis at the level of the fifth thoracic segment, with fecal and urinary incontinence. She remained practically in this condition for seventeen months, when she was admitted to the Montefiore Hospital in a bedridden condition on Jan. 29, 1928. She had a paraplegia of the upper motor neuron type, with bilateral pes equinovarus and spontaneous flexion and extension movements of both lower extremities in which the muscles were atrophied. There was complete sensory paralysis from the toes to the fifth dorsal segment, with loss of bladder and rectal control.

Laboratory Data.—The spinal fluid was normal. Roentgen examination of the spine disclosed a crushed fracture of the fourth dorsal vertebra.

Course.—The neurologic condition remained unchanged until February 23, when decubitus ulcers developed; the patient died of sepsis on June 24.

The clinical and anatomic diagnosis was fracture of the vertebra; transverse myelitis; rheumatic endocarditis of the mitral valve, with mitral stenosis and insufficiency; chronic passive congestion of the liver; chronic purulent cystitis; renal

calculus; multiple abscesses of the kidneys; pulmonary emphysema; multiple extensive decubitus ulcers.

Autopsy.—Gross Examination: Sections from the upper thoracic to the sacral region of the cord alone were available for study. The cord between the sixth and eighth dorsal segments was thin; most of its fiber tracts were translucent.

Microscopic Examination: A cross-section from the fifth dorsal segment showed distortion of the cord, with complete destruction of all fiber tracts (fig. 5*A*). At the sixth dorsal segment all fiber tracts, except those of the posterior and part of the left anterolateral columns, were completely demyelinated

Fig. 4.—*A*, complete destruction of axis cylinders in the hemorrhagic areas. Bielschowsky stain; reduced from × 480. *B*, destruction and swelling of axis cylinders near the hemorrhagic area; most of the cylinders, however, are fairly well preserved. Bielschowsky stain; reduced from × 480.

(fig. 5*B*). Small necrotic foci were present throughout all segments (fig. 6). The meninges were thickened and contained psammoma bodies and vessels with narrow lumens. The meninges from the ventral portion of the cord, below the fifth dorsal segment, contained accumulations of round cells. The anterior spinal artery was fairly normal; the remaining vessels of the cord were moderately arteriosclerotic, but were free from adventitial infiltration. The demyelinated portion of the cord had a slightly honeycombed appearance and was filled with compound granular corpuscles, some of which were loaded with fat. With the cresyl violet stain the

anterior horn cells presented various degrees of degeneration, shrinkage and loss of Nissl substance. Sections of the cord below the sixth dorsal segment showed only descending degeneration and necrobiotic foci. Under a higher magnification, broken-down thickened fiber tracts and numerous compound granular corpuscles were seen. The axis cylinders were completely fragmented and appeared granular. Both

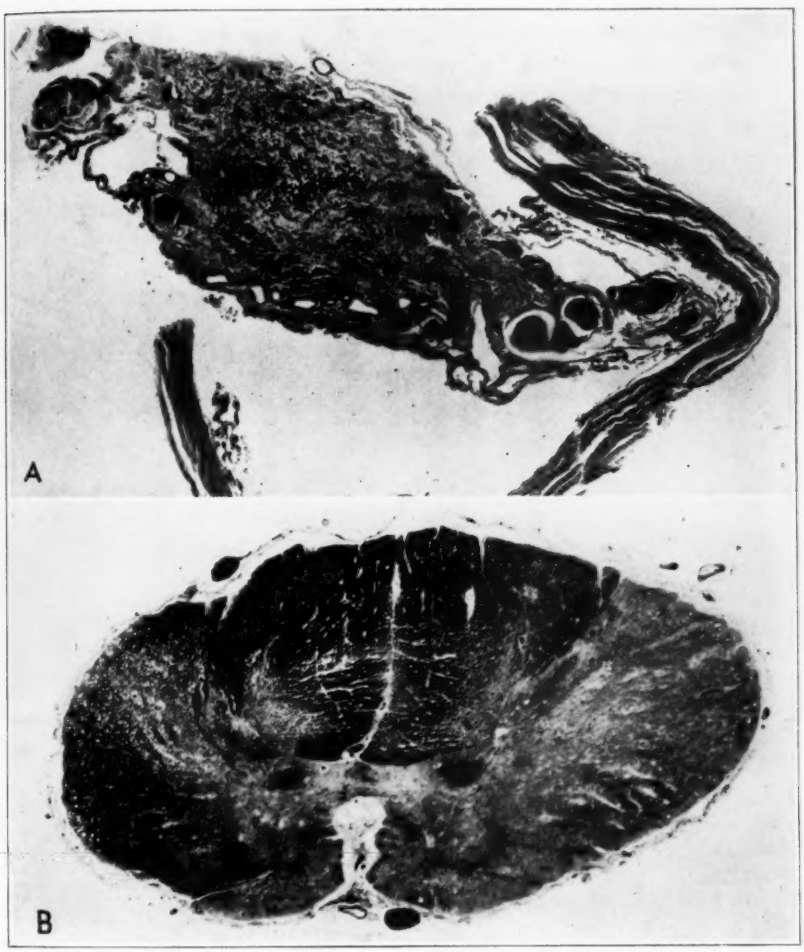

Fig. 5.—*A*, transverse section of the fifth thoracic segment, showing distortion of its outline with complete destruction of all the fiber tracts. Myelin sheath stain (Weil modification). *B*, section below *A*, showing slight sparing of the posterior and part of the left anterolateral columns; all the other tracts are demyelinated. Myelin sheath stain (Weil modification).

regressive and progressive glial changes were observed in the destroyed area (fig. 7).

The microscopic diagnosis was myelopathy, traumatic; concussion.

Comment.—In this case a transverse lesion of the cord developed following a fractured vertebra. It differs from case 1 by the absence of evidences of hemorrhage or of direct injury to the cord. It is impossible to state on histologic grounds what the precise nature of the lesion was immediately after the injury. There were no evidences of inflammation, the changes in the cord resembling those in compressions. The posterior columns below the area of compression showed necrotic foci of the type observed in concussion of the spinal cord. A study of this case in conjunction with case 1 would seem to suggest that the time element plays as important a rôle in determining the nature of the pathologic process as does the severity of the injury.

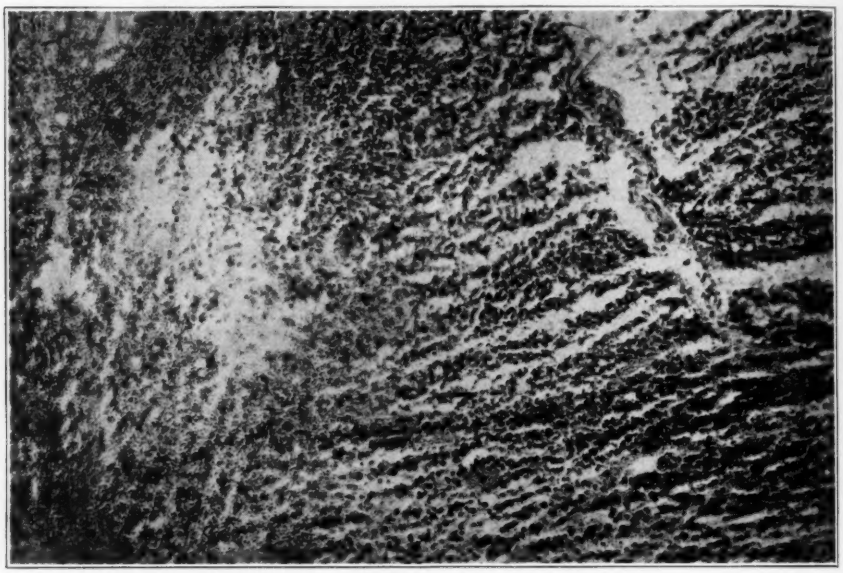

Fig. 6.—Small necrotic focus. Such foci were present throughout the cord. Myelin sheath stain (Weil modification).

Case 3.—L. H., a boy, aged 17, was thrown forcibly to the ground in an automobile accident in June, 1927. After regaining consciousness he was unable to move his lower extremities and had loss of sensation from the toes to the umbilicus. He was admitted to the Jewish Hospital of Brooklyn on September 1. There, neurologic examination revealed evidences of an incomplete transverse lesion of the cord at the seventh cervical segment and a partial Horner's syndrome on the right side. The patient was unable to raise his head and shoulders and was incontinent of urine and feces. Neurologic examination on admission to the Montefiore Hospital, on Jan. 24, 1928, gave practically the same results, except that the level had ascended to the fourth cervical segment.

Laboratory Data.—Roentgenologic examination disclosed partial absorption and a mottled appearance of the seventh cervical vertebra, possibly an old fracture. Except for pyuria, other laboratory data were normal.

Course.—The condition remained stationary until a few months before death, when a severe cystitis developed, with an irregular fever. On Nov. 4, 1931, the patient became stuporous. The urea nitrogen, which hitherto had remained within normal limits, was 100.7 mg. He died on November 14.

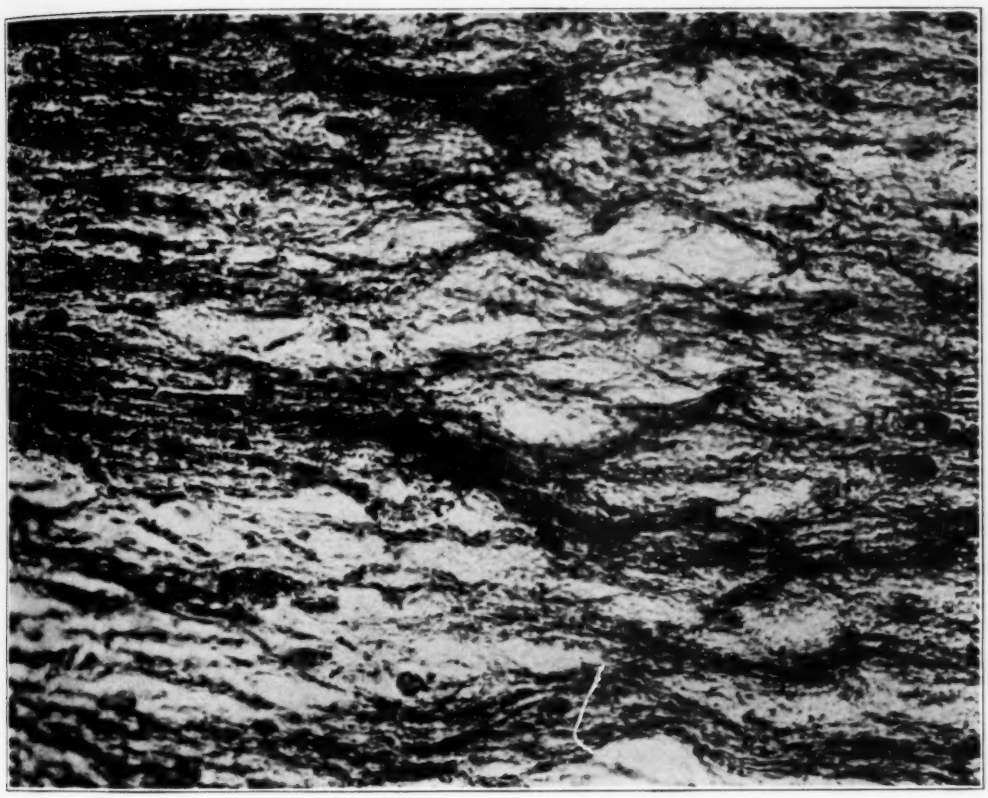

Fig. 7.—Longitudinal section, showing progressive and regressive glial changes in the destroyed areas. Mallory phosphotungstic stain; × 400.

The clinical and anatomic diagnosis was fracture of the cervical spine; transverse myelitis; transverse myelopathy, traumatic; acute and chronic cystitis; ureteritis and pyelonephritis; bronchopneumonia of the lower lobe of the left lung.

Autopsy.—Gross Examination: The lower cervical region of the spinal cord was constricted, and the dura in this region was thickened. The posterior columns at the fourth cervical segment were translucent. The substance of the cord was destroyed in the constricted area.

Microscopic Examination: Sections from the fourth cervical to the upper dorsal segments of the spinal cord showed a slightly thickened dura, compression of the

vessels with narrowed lumens and complete demyelinization and distortion of almost all fiber tracts (fig. 8). The cervical segment above the constricted portion of the cord disclosed ascending demyelinization of the posterior columns and of the cerebellar pathways. The segments at the area of constriction showed a complete destruction of all pathways (fig. 8); these were replaced by compound granular corpuscles, fragmented myelin sheaths and, in places, attempts at repair by connective tissue and proliferation of the blood vessels. No inflammatory cells were

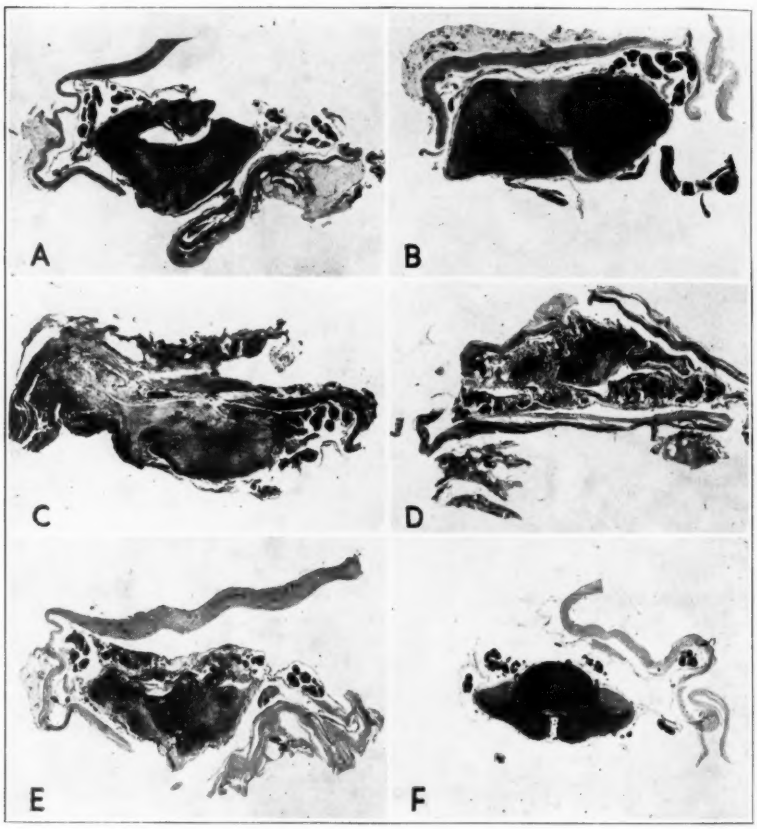

Fig. 8.—*A* and *B*, transverse sections of the cervical segments above the constricted areas, showing ascending demyelinization. *C*, *D* and *E*, segments of the cord in the region of constriction, showing complete destruction of all fiber tracts. *F*, segment of the cord below the traumatized region, showing descending demyelinization.

observed. In cresyl violet preparations some of the anterior horn cells were destroyed; others showed marked swelling, complete disappearance of the Nissl substance and large pale eccentric nuclei with deeply stained nucleoli. The lower thoracic segments presented a descending demyelinization of the crossed pyramidal tracts. The myelin sheaths in the destroyed areas were markedly disintegrated;

the axis cylinders were completely gone, and there was little attempt at glial repair. Small areas of necrosis were found in the cord above and below the constricted area.

The microscopic diagnosis was myelopathy, traumatic.

Comment.—In this case, as in case 2, there was transverse myelopathy following a fractured vertebra. Here again necrotic foci of the type observed in concussions of the spinal cord were found. There was no evidence of any inflammatory reaction in any part of the cord.

CASE 4.—B. P., a man, aged 54, was admitted to the hospital on March 22, 1930, with the history that in September, 1929, he had had pain in both shoulders. Within two weeks, the pain extended to the arms; it was aggravated by coughing, sneezing and straining; six weeks later it radiated down the spine. Following a diagnosis of Pott's disease, he was placed in a plaster of paris cast. About three months before admission, while the cast was being removed, the patient experienced severe pain in the back of the neck; this was followed immediately by quadriplegia with loss of sphincteric control.

Examination.—On admission physical examination revealed evidences of an old quiescent pulmonary tuberculosis and tenderness over the sixth and seventh cervical vertebrae, with crepitation over the latter.

Neurologic examination showed Horner's syndrome on the right and a horizontal nystagmus in both eyes. There was a quadriplegia, with amyotrophy of the upper extremities; the paralysis was more marked in the lower extremities. The reflexes were diminished in the upper extremities and exaggerated in the lower. There were a Babinski sign on the right and toe drop on the left. Sensory examination revealed a belt of hyperalgesia at the fifth cervical segment, with complete loss of all forms of sensation below this level.

Laboratory Data.—Roentgenologic examination revealed apical fibrous tuberculosis; the fifth and sixth cervical vertebrae appeared moth-eaten, with crushing of the intervertebral disks. The roentgenologic findings were reported as suggestive of tuberculosis rather than of a malignant process. Manometric studies of the spinal fluid showed an almost complete subarachnoid block. The spinal fluid contained total protein, 180 mg., and increased globulin and albumin. The sputum contained tubercle bacilli.

Course.—The patient had no fever, but complained of severe pain in the back of the neck which required opiates for relief. He died suddenly on April 5, 1930.

The clinical and anatomic diagnosis was tuberculous osteomyelitis of the cervical vertebrae; compression myelitis of the spinal cord; bilateral fibroid pulmonary tuberculosis; thrombosis of the right coronary artery; healed infarct of the heart; cardiac hypertrophy and dilatation; hypostatic bronchopneumonia.

Autopsy.—Gross Examination: There was an area of marked compression of the cord at the level of the fifth and sixth cervical vertebrae. The bodies of these two vertebrae appeared brittle and projected into the spinal canal; the adjacent tissues contained a pocket of thick white pus, which extended to the apex of the right lung but did not communicate with it or with the pleural cavity. Corresponding to the lesion in the spinal column, the cord showed several constrictions between the third and seventh cervical segments. At the sixth and seventh cervical segments the cord was completely crushed, so that its structure could barely be distingushed.

Microscopic Examination: Sections of the medulla oblongata revealed slight distortion in the region of the tenth and twelfth cranial nerve nuclei. One of the

pyramids showed mild destruction of some of the myelin sheaths. The meninges on the same side were somewhat thickened, and the vessels showed signs of compression and narrowed lumens.

The constricted area at the third cervical segment showed distortion of the cord and destruction of the right third of its dorsolateral portion (fig. 9 *A*). The columns of Goll, Lissauer's tract and part of the crossed pyramidal tracts on that side were completely destroyed. The anterior columns on the left side were partially demyelinated. Under a higher magnification, some of the myelin sheaths

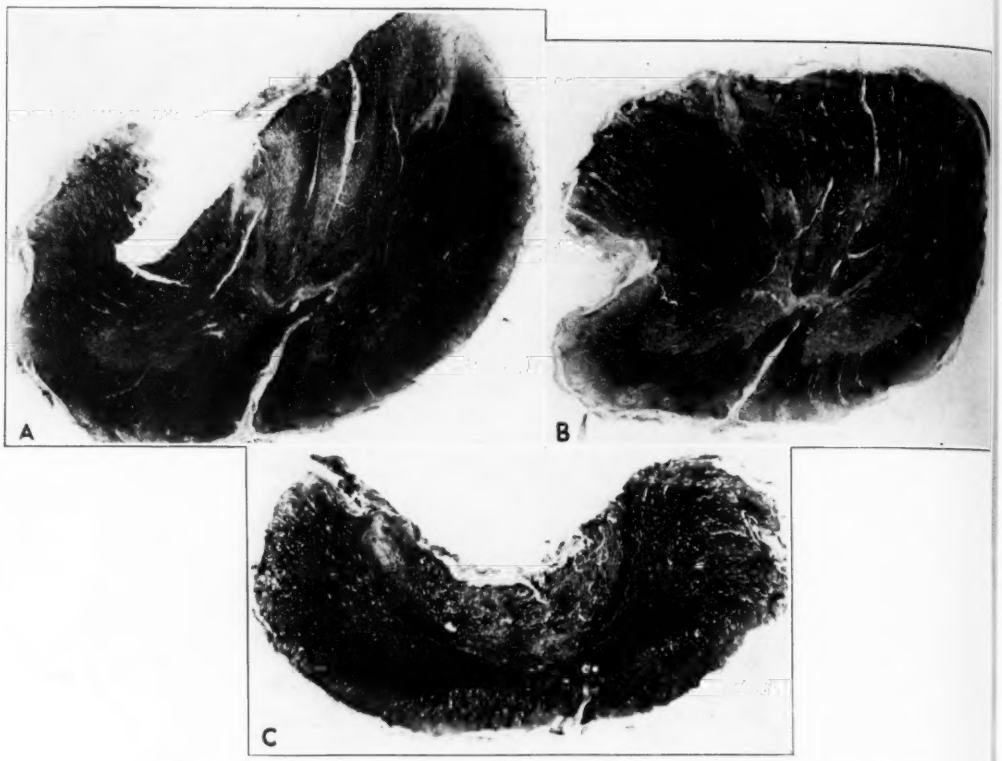

Fig. 9.—*A*, transverse section of the third cervical segment, from the upper level of constriction, showing destruction of the right dorsolateral portion of the cord. *B*, transverse section of the sixth cervical segment, the level of the second constriction, showing destruction of right lateral part of the cord and partial demyelinization of the anterior and posterior columns. *C*, transverse section of the seventh cervical segment, the level of the third constriction, showing destruction of the dorsal part of the cord. Notice the sievelike appearance of the cord. Myelin sheath stain (Weil modification).

appeared disintegrated, while others were well preserved. The meninges were slightly thickened, and the anterior spinal vessels were compressed, as evidenced by their narrow lumens. The ganglion cells in the right dorsal gray column were completely destroyed, but those of the ventral horns were fairly well preserved

on both sides. Sections between the medulla oblongata and the third cervical segment showed slight distortion of the cord, ascending degeneration, somewhat thickened meninges and compression of the intraspinal vessels.

At the level of the second constriction (between the third and seventh cervical segments) the right posterior horn and the dorsocerebellar tracts appeared destroyed. At the fifth cervical segment the cord was fairly intact, showing only demyelinization of the posterior columns and slight pallor of the crossed pyramidal tracts. At the sixth cervical segment there was a wedge-shaped area within the right lateral pyramidal tract, with partial demyelinization of the anterior and posterior columns (fig. 9 *B*). The meninges were somewhat thickened, and a few of the anterior horn cells showed pigment atrophy and granular degeneration. At the level of the third constriction (the seventh cervical segment) there was complete destruction of the dorsal third of the cord (fig. 9 *C*). The remainder of the cord had a sievelike appearance, which, under a higher power lens, showed marked destruction of some of the myelin sheaths, with swelling of others. The gray matter was markedly compressed and could hardly be distinguished under a low power lens. The meninges were thickened and compressed the vessels. The anterior horn cells had undergone various stages of destruction, with loss of Nissl substance. In most ganglion cells neither nucleus nor nucleolus could be seen. There were no evidences of inflammation anywhere. All sections below this level showed a descending demyelinization. Longitudinal sections in the constricted areas showed, with the sudan IV stain, accumulations of compound granular corpuscles. Myelin sheath preparations revealed destruction of some of the myelin sheaths, with preservation of a few and swelling of others. Sections stained with the Bielschowsky method disclosed only a few axis cylinders remaining in the destroyed areas; these were fragmented and had a corkscrew appearance and ball-like terminations. With the Victoria blue stain there was a poor glial response; in some areas there was an attempt at glial proliferation in a disorderly fashion.

The microscopic diagnosis was myelopathy, traumatic; crushed vertebra.

Comment.—The absence of evidences of an inflammatory process throughout the meninges and cord excludes infection as the cause of the pathologic process. This case belongs with the group of cases of direct compression of the spinal cord by fractured vertebrae with mild or moderate vascular interference.

Case 5.—F. B., a man, aged 57, was admitted to the hospital on Oct. 22, 1920, complaining of weakness and pain in the back. In March, 1918, he had had a cold abscess in the upper part of the sternum. The affected bone was resected, and the patient remained well for six months. In the early part of 1919, he had another cold abscess in the dorsolumbar region, which was also incised and drained.

Examination.—Physical examination revealed bilateral pulmonary tuberculosis.

Neurologic examination revealed a spastic paraplegia, with hyperreflexia and a bilateral Babinski sign, and amyotrophy of the lower extremities. There was a large painless superficial burn over the anterior surface of the upper part of the right thigh; there was a belt of hyperalgesia at the eighth dorsal segment, below which there was total analgesia on the right side. On the left there was an area of hypalgesia from the eighth to the twelfth dorsal segment, and below the twelfth dorsal segment there was analgesia. The sense of touch was lost below the latter segment on both sides.

Laboratory Data.—Roentgenologic examination showed diffuse rarefaction in the upper portions of each humerus and destruction of the vertebral portions of the fourth and fifth ribs on the left.

Course.—On Jan. 15, 1921, the patient had retention of urine, which was followed by incontinence. On February 3, there appeared bed sores, and on May 26, paraplegia in flexion. Death occurred from bronchopneumonia on May 29.

The clinical and anatomic diagnosis was tuberculous spondylitis of the middorsal spine; compression myelitis; Pott's disease involving the second and third thoracic vertebrae, with destruction of the spinous process of the third and articulating heads of the third, fourth and fifth ribs of the left side; bronchopneumonia.

Autopsy.—Gross Examination: There were a gibbus in the upper thoracic region and a slight scoliosis in the lumbar region. A specimen of the spinal column from the sixth cervical to the sixth thoracic vertebra, with a portion of the spinal cord included in this region, was received for examination. A section through the center of the specimen disclosed a cavity formed by destruction of the adjacent arches of the second and third thoracic vertebrae. The cavity was traced into a sinus which had eroded the third, fourth and fifth ribs obliquely. Posteriorly, there was destruction of the spinous process of the third thoracic vertebra, so that the wall of the spinal canal and its dura hung almost free in the posterior part of the cavity, the walls of which consisted of roughened necrotic tuberculous granulation tissue.

Microscopic Examination: With the myelin sheath stain, sections of the cervical region showed ascending degeneration of the column of Goll, the column of Burdach being uninvolved; the dorsocerebellar tracts also showed slight demyelinization. Sections of the cord at about the eighth dorsal segment showed demyelinization of the crossed pyramidal tracts. With the hematoxylin-eosin stain, the meninges appeared slightly thickened and contained small accumulations of inflammatory cells; the vessels were also somewhat thickened. With the Nissl stain, the anterior horn cells were normal in shape and size although the Nissl substance stained poorly. These cell changes were observed in the cervical as well as in the thoracic portions of the cord. Longitudinal sections with the Mallory phosphotungstic stain showed isomorphous gliosis in the demyelinated areas.

The microscopic diagnosis was myelopathy; trauma; Pott's disease.

Comment.—The gross appearance of the cord and the microscopic evidences of an ascending and descending degeneration leave no doubt that the condition is a myelopathic process following destruction of the vertebrae, with secondary compression of the cord and its vessels. In contrast to case 2 of the infectious group, in which a tuberculoma invaded the spinal cord and caused its destruction, this case was one of a myelopathic process due to direct compression of the cord and its vessels.

GENERAL COMMENT

Five cases of myelopathy due to trauma have been studied. In case 1 there was a lesion of the cord due to a tear of the meninges and direct injury of the cord by a bullet. The histologic picture at the level of the injury was one of hemorrhage in the substance of the cord. There were also inflammatory changes in the meninges and about the vessels of the cord. It is well known that in injuries of any tissue there

occurs an immediate migration of phagocytic cells to the site of injury in order to remove the products of disintegration. Although inflammation in nerve tissue does not necessarily always indicate infection, there is nevertheless an intimate relationship between the two, a view that has already been emphasized by one of us (Davison).[1] The possibility that some of the inflammatory cells may have been due in this case to secondary invasion from the infected wound at the site of the trauma cannot be denied.

Schmaus [2] produced direct injuries of the cord by cutting or piercing it with a needle in dogs and rabbits. He also found migration of inflammatory cells to the site of injury. The histologic picture in the cords of these animals was that of a combination of a myelopathic and a myelitic process, with predominance of the former. Changes in the spinal cord due to direct trauma may present a histologic picture resembling infection (myelitis) or destruction of the cord of the type observed in softening (myelopathy). Such lesions may be associated with small or extensive hemorrhages as a result of direct injury to the vessels of the substance of the cord, as well as with pseudoheterotopia from lacerations of the cord.

Cases 2 and 3 belong with a group of cases in which trauma of the vertebral column produces fracture of one or several vertebrae; the bone fragments, torn ligaments and the usually associated extradural and intradural hemorrhages exert pressure on the cord directly, and damage it by interfering with its circulation. The patients in cases 2 and 3 lived two and one-half and four and one-half years after the injury, respectively, so that the process was not as acute as in case 1. There was no evidence of inflammation, the lesions resembling more those observed in a myelopathic process from compression and circulatory interference. The blood supply in both cases, however, was not sufficiently interfered with to produce the usual picture of complete softening of the cord due to vascular disease from other causes. The slight glial response and increase in protoplasmic astrocytes would seem to indicate a chronic process similar to that in some cases of compression from tumor. In these cases there were also focal areas of necrosis such as have been described in concussions of the spinal cord. Lesions of this type are more common in the brain (commotio cerebri). This is due to the fact that the brain is enclosed in a rigid, practically unyield-

1. Winkelman, N. W., and Davison, C.: Subacute Combined Degeneration of the Spinal Cord: A Study of the Underlying Pathologic Process, Arch. Neurol. & Psychiat. **29**:317 (Feb.) 1933.

2. Schmaus, H.: Beiträge zur pathologischen Anatomie der Rückenmarkserschütterung, Virchows Arch. f. path. Anat. **122**:326, 1890. Schmaus, H., and Sacki, S.: Vorlesungen über die pathologische Anatomie des Rückenmarks, Munich, J. F. Bergmann, 1901.

ing receptacle, in contrast to the spinal cord, which, owing to greater free space between it and the vertebral column, is much more mobile than the brain. Schmaus [2] traumatized the spinal cords of dogs and rabbits by directing blows to wooden planks against which the vertebral columns of animals rested. According to him, the lesions in the cords of these animals were caused by the blow to the vertebral column, which transmitted an impulse to the cerebrospinal fluid and produced pressure on the spinal cord; this transmitted pressure ruptured the walls of the blood vessels and gave rise to small or large hemorrhages, depending on the size of the vessel injured. When the hemorrhages were sufficiently small to permit absorption, the animals recovered. In some cords anemic softenings as well as small necrotic foci were found.

In cases of shell explosions, Claude and Lhermitte [3] found changes in the cord, such as primary degeneration of the fiber tracts, excessive hypertrophy of the axons, swelling of the myelin, formation of myelophages and neuroglial reactive phenomena. Mairet and Durante [4] reported medullary concussions in animals induced by vibration of the air following the use of high explosives. Guillain [5] maintained that a number of his cases of concussion were due to air pressure changes produced by the force of the explosion. As Hassin [6] and others have pointed out, the essential cause of the pathologic changes is the concussion itself.

It would seem that a number of the cases diagnosed as functional disease of the spinal cord, so-called "railway spine," in which the patients received blows and severe vibrations (following explosions and similar accidents) to the vertebral column and later developed symptoms referable to the spinal cord, might be included in this group.

Cases 4 and 5 illustrate the effect of a tuberculous process in the vertebral column on the spinal cord. Whether in case 4 the sudden onset of a quadriplegia with rapid progression of symptoms following the removal of the cast was a mere coincidence, or whether this procedure added additional trauma to the preexisting lesion in the cord is difficult to state. At any rate, these cases both illustrate that a tuberculous lesion in the vertebrae may produce a myelopathy by direct pressure on the cord, as well as on its vessels, by giving rise to circulatory interferences.

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3. Claude and Lhermitte: Etude clinique et anatomo-pathologique de la commotion médullaire direct par projectiles de guerre, Ann. de méd. **2**:479, 1915.

4. Mairet, A., and Durante, G.: Contribution à l'étude expérimentale de lésions commotionnelles, Presse méd. **25**:478, 1917; Rev. neurol. **26**:97, 1919.

5. Guillain, G.: Travaux neurologiques de guerre, Paris, Masson & Cie, 1920, p. 307.

6. Hassin, G. B.: Concussion of the Spinal Cord, Arch. Neurol. & Psychiat. **10**:194 (Aug.) 1923.

SUMMARY

1. Five cases are presented to illustrate lesions of the spinal cord following trauma.

2. In case 1, with direct trauma of the cord, the lesion was predominantly myelopathic, with some slight evidences of inflammation. The latter is most likely only a secondary reaction to the trauma. In cases 2 and 3, the pathologic process was also essentially myelopathic but was due to compression of the cord by fractured vertebrae, with secondary circulatory interference and concussion below the level of the injury.

3. Cases 4 and 5 illustrate myelopathic processes of the spinal cord due to a tuberculous lesion in the vertebrae causing direct pressure on the cord, as well as on the vessels, and leading to circulatory interference.

THROMBOSIS OF INTRACRANIAL ARTERIES

REPORT OF THREE CASES INVOLVING, RESPECTIVELY, THE ANTERIOR CEREBRAL, BASILAR AND INTERNAL CAROTID ARTERIES

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By far the greater proportion of cases of cerebral thrombosis occur in the middle cerebral artery or its branches, resulting in the well known hemiplegia, with or without aphasia. Owing to the infrequency with which primary thrombosis of cerebral arteries other than the middle cerebral is encountered, the associated clinical syndromes are not generally familiar and accordingly may sometimes pass unrecognized. In the following account three cases of cerebral thrombosis are described. Each case illustrates well the syndrome resulting from occlusion of the particular artery at fault. The cases are of interest from a pathologic standpoint also, and a report of the postmortem observations is included.

REPORT OF CASES

CASE 1.—*Thrombosis of the anterior cerebral artery (congenital anomaly).*

History.—Mrs. G. M., aged 61, a scrubwoman, who was admitted to the Toronto General Hospital on May 3, 1932, had been well until two years previously when she suddenly lost consciousness while working. She recovered consciousness in a short time, but was unable to speak and failed to recognize people for about two days. Her condition gradually improved and after a few weeks' rest she resumed work. Friends noticed that her memory was mildly impaired after this, and that on occasions she talked foolishly and showed poor judgment. She continued working until five days before admission to the hospital, when she awakened in the morning feeling "dizzy." Later in the day, while at work, she "went weak all over" and collapsed on the floor. When discovered, she was conscious and could talk but was unable to stand. She was taken home and remained quietly in bed for three days. Subsequently she became talkative and profane. Weakness of the left arm was noted. It was decided to bring her to the hospital.

Examination.—The patient was euphoric and talked incessantly. Her theme was almost entirely sexual. She addressed the examiner with exaggerated terms of endearment, interspersing her remarks with coy smiles and winks. She continually begged to be kissed and attempted to kiss the examiner. At times she exposed her body, making vulgar allusions. It was difficult to hold her attention sufficiently to make an adequate mental examination. She was well oriented for place and fairly well for time. Her knowledge of current events was limited. She could do simple problems in addition and multiplication with fair accuracy.

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Replies to questions regarding her past life were usually facetious, but several statements (later corroborated) showed a fair recollection of the principal events. Memory for recent events was inaccurate but not grossly impaired. She could name objects correctly and could understand written speech.

No abnormalities were detected in the cranial nerves. The retinal arteries showed moderate arteriosclerotic changes. Several small fresh hemorrhages were seen along the course of the vessels in the right retina close to the disk and extending out toward the periphery. No hemorrhages were present in the left fundus. The right arm was in a constant state of purposeful activity, grasping at anything that came within reach. The limb required to be forcibly held down to permit examination; otherwise she would grasp the head, coat or hand of the examiner. If nobody was near her, she would grasp the bedclothes, the end of the bed or any convenient object. Anything she grasped would be released on command (incomplete grasp reflex). The right leg moved restlessly, chiefly in flexion and extension at the knee. If commanded she could stop the movements of the arm and leg, but after a very short interval they would recommence. In the right arm and leg power was good at all joints. Tone and coordination were normal. The left arm and leg lay immobile in extension. If told to make a specific movement with the left upper limb she was totally unable to do so (apraxia). When any object was placed in the left hand it was gripped tightly and force had to be used to release it. If the object was left in the hand without any attempt to release it for two or three minutes, relaxation occurred spontaneously. Attempts to gain release shortly after the object was grasped resulted in an increase in the grip (grasp reflex). The muscles of the left shoulder and elbow were flaccid; the patient could not voluntarily move the limb and could offer only mild resistance to passive movements at these joints. Weakness was greater at the shoulder than at the elbow. There was no power in the left leg at any joint; tone was slightly increased. The reflexes in the arms were moderately active, with no difference on the two sides. The left knee jerk was more active than the right. The abdominal reflexes were not elicited. Plantar flexion occurred on the right; dorsiflexion, on the left. No clonus was present. A pinpoint was appreciated everywhere, with no definite difference on the two sides of the body. Light touch, vibration sense, sense of position and passive movement and two point discrimination could not be tested accurately owing to the defect in attention, but no gross impairment was detected. Stereognostic sense was good in both hands. There was no sphincter disturbance at this time.

The peripheral vessels were thickened and slightly tortuous. The blood pressure was 170 systolic and 110 diastolic. The heart was moderately enlarged; the cardiac rate was 68; the rhythm was regular; the sounds were of fair quality; there were no murmurs. The lungs and abdomen were normal.

The cerebrospinal fluid was clear and was under a pressure of 190 mm. There were 20 mononuclears per cubic millimeter; the Pandy and the Wassermann tests were negative. The urine was normal. The Wassermann test of the blood was negative.

Diagnosis.—The diagnosis was thrombosis of the right anterior cerebral artery.

Course.—Three days after admission a marked change occurred. The patient lay quietly in bed with her eyes closed. She made no spontaneous utterance. If addressed she would open her eyes and smile faintly, but would not answer except occasionally to mutter something incomprehensible. She took nourishment but required to be fed. She appeared to understand some things that were said to her. The left arm, previously immobile, was constantly on the move, grasping

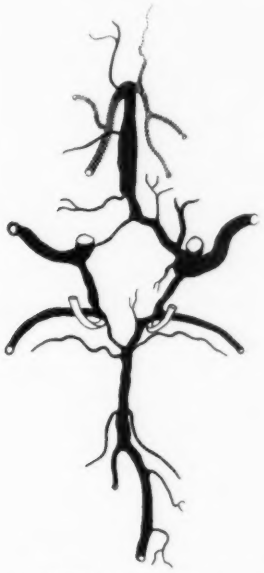
and groping at objects within reach. The grasp reflex was still present. The right arm lay flaccid in extension, apparently without power at any joint; no evidence of a grasp reflex was present. The left biceps jerk was more active than the right. The left leg, which formerly had been totally paralyzed, now weakly flexed at the knee when the sole was stimulated. The right leg was apparently without power except for a very slight withdrawal when stimulated. Tone was increased in the left leg, and the left knee jerk was still more active than the right. Both plantar reflexes were in extension. She was incontinent of urine and feces. After this condition had persisted for about twenty-four hours, movement of the left arm ceased and the patient lapsed into coma. Her temperature rose, and death occurred on the seventh day after admission.

Postmortem Examination.—The brain showed nothing remarkable on gross examination apart from advanced arteriosclerosis of the cerebral arteries. The only palpable or visible area of softening was a small area underlying the ependyma at the tip of the frontal horn of the right lateral ventricle. The right anterior cerebral artery was a threadlike structure which joined the normal-sized left anterior cerebral to form a common trunk. Immediately beyond the junction was the commencement of a thrombus which continued until the vessel had passed over the genu of the corpus callosum. On the dorsal surface of the genu, just behind its tip, the artery divided into two branches which were distributed in the normal manner on the mesial surfaces of both hemispheres. Branches were given off to the frontal lobes as follows: two branches from the left anterior cerebral artery, before its junction with the right, to supply the anterior inferior portion of the left frontal lobe; two small branches from the right side of the common trunk to supply the anterior inferior part of the right frontal lobe; a branch from the common trunk, as it rounded the genu of the corpus callosum, to supply the anterior superior portion of the right frontal lobe; a branch at the point where the artery divided, to supply the left frontal lobe in its anterior superior part (fig.). The remaining arteries of the circle of Willis were abnormally distributed in several respects: The right vertebral artery was much smaller than the left; the right posterior cerebral artery commenced as a very small artery from the basilar, but at the junction with the posterior communicating artery it became the equal in caliber of the left posterior cerebral artery. The internal carotid and middle cerebral arteries were not abnormal in size or distribution.

Microscopic examination of the thrombosed artery revealed severe endarteritis, with marked fibrous thickening of the tissue internal to the internal elastic lamina. The medial coat was not markedly altered; the adventitia had a number of lymphocytes in its substance. The lumen of the vessel contained a thrombus in an early stage of organization but was not completely occluded. Examination of the brain revealed acute degenerative changes in the cortex and white matter on the medial surfaces of both frontal lobes. The cortical cells showed varying degrees of chromatolysis. Destruction of myelin was most marked in the region of the anterior horns of both ventricles. Sections from the paracentral lobules (leg areas) of both hemispheres showed less severe but marked breakdown of myelin. The acute degenerative changes were more severe in the right frontal lobe than in the left. In the latter there were some signs of reaction with fibroblasts and astrocytes.

Summary.—A scrubwoman, aged 61, five days before admission to the hospital collapsed while working. She experienced no loss of consciousness, but was unable to stand. Three days later she complained

of weakness in the left arm. On admission, she was euphoric and loquacious and at times became vulgar and profane. Attention was poor, but there was no gross mental confusion. Fresh hemorrhages were present in the right retina. The left arm and leg lay immobile in extension; the left leg was completely paralyzed; the left arm was partially paralyzed, with apraxia and a grasp reflex present. The right arm and leg were in a constant state of motor activity, the right hand grasping objects within reach but releasing them on command. A few days later she became aphasic; the right arm and leg were totally paralyzed; the left arm, formerly immobile, moved, constantly groping



Drawing of the circle of Willis (case 1) showing anomalous arterial distribution.

at objects. There was some return of power in the left leg. Both plantar reflexes were in extension. Subsequently the patient became comatose, and all movement in the limbs ceased. Death occurred on the twelfth day after the onset.

Post mortem, an anomalous distribution of the anterior cerebral arteries was found. They joined to form one long stem which subsequently divided into two on the dorsum of the corpus callosum. A thrombus was present in the common trunk.

Comment.—The syndrome which results from occlusion of one anterior cerebral artery is well known. According to Critchley,[1] if the

1. Critchley, M.: Brain **53**:120, 1930.

thrombus commences above the junction of the anterior communicating artery with the main trunk the principal symptoms will be: (1) paralysis of the opposite lower limb due to ischemia of the cortical leg area; (2) ideomotor apraxia affecting the left arm, whether the limb is ipsilateral or contralateral, as a result of softening of the anterior part of the corpus callosum; (3) psychomotor disturbances in the upper limb of the same side as the paralyzed leg (these comprise forced grasping and groping [grasp reflex] and are the result of release of primitive automatic centers deprived of controlling influences from higher centers in the frontal lobe cortex [Adie and Critchley][2]).

When this patient first came under observation, the symptoms suggested thrombosis of the right anterior cerebral artery—complete paralysis of the left leg and partial paralysis of the left arm, with apraxia and a grasp reflex. Additional findings, which could not be entirely explained by a lesion in this situation, were the mental status and the persistent motor activity of the right arm and leg. The continuous grasping of the right hand was under voluntary control, and objects grasped could be released at will. Such a phenomenon is called an incomplete grasp reflex to distinguish it from the grasp reflex in which voluntary release of objects is impossible. It was probably the result of a lesion in the left frontal lobe similar to but much milder than that on the right side. The mental reaction with euphoria and freedom from inhibitions resembled what is sometimes seen in dementia paralytica. It did not appear until the third day after the onset of the illness, and one may reasonably assume that it was the result of the bilateral lesion rather than of any general circulatory disturbance. Definite evidence of the bilateral nature of the lesion was afforded when paralysis of the right leg and arm occurred. At this time some improvement in the power of the left arm and leg was noted.

The explanation of the sequence of events from the anatomic standpoint is not entirely satisfactory. The left frontal lobe received blood from two branches arising from the left anterior cerebral artery before it united with the right anterior cerebral to form a common trunk. The right anterior cerebral artery had no branches corresponding to these, and the right frontal lobe derived all of its blood supply from branches emerging from the common trunk. It is probable that when partial occlusion first occurred, the two branches enabled the left frontal lobe to receive a more adequate blood supply than the right. Later, the thrombus having increased, the flow through the main trunk was further diminished and the circulation to the left frontal lobe was inadequate. The improvement in power of the left leg and arm, which occurred at the same time as the onset of paralysis on the right side, suggests that

2. Adie, W. J., and Critchley, M.: Brain **50**:142, 1927.

blood formerly going to the left frontal lobe was diverted to flow to the right. This was only temporary, however, as on the following day coma and paralysis of all four limbs developed.

Bilateral thrombosis of the anterior cerebral arteries occurs rarely; usually it is found post mortem associated with areas of softening elsewhere. Schuster [3] recorded the case of a man, aged 73, who suffered two strokes within four years. He became paralyzed in both legs and demented. He was completely disoriented and made no spontaneous utterance. Examination showed weakness of the right corner of the mouth. The right hand moved restlessly, clutching at the bedclothes and any objects within reach; force had to be used to release the objects. The left forearm was held tightly across the chest, with the fist clenched. Both legs were very weak, and there was an increase in tone of the left leg. The tendon jerks were increased, especially on the left side. The plantar responses were in extension. At autopsy there was bilateral thrombosis of the anterior cerebral arteries.

Critchley [1] described a case of thrombosis of the left anterior cerebral artery occurring in a man in whom, owing to a congential peculiarity, this vessel supplied both hemispheres. Eleven months before coming under observation the patient, aged 45, had weakness in the legs. The principal findings on admission to the hospital were a spastic paraplegia in the lower limbs and some increase in tone in the arms, with a distinct catatonic tendency. About ten days after admission, he suddenly cried out with intense headache, vomited and lost consciousness. The arms became very spastic. Coma deepened, and he died four days later. Autopsy showed advanced cerebral arteriosclerosis and a quantity of free blood in the subdural space. The right anterior cerebral artery was very small and supplied only the most anterior portion of the frontal pole. At the level of the genu the left anterior cerebral artery bifurcated and sent one branch across the middle line so as to supply the mesial aspect of the right hemisphere. In this way the left anterior cerebral artery was supplying the cortex, including the paracentral lobules, of both halves of the brain. A thrombus of the first part of the main trunk of the left anterior cerebral artery was present, causing ischemia of the cortical areas corresponding with both lower limbs.

These two cases contrast with the case described here, as the patients survived the initial attack, first coming under observation when considerable time had elapsed. Bilateral motor paralysis of the lower limbs was a feature in all three cases; this finding, whether unilateral or bilateral, is the most important physical sign in lesions of the anterior cerebral artery.

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3. Schuster, P.: Ztschr. f. d. ges. Neurol. u. Psychiat. **83**:586, 1923.

The congenital anomaly whereby the two anterior cerebral arteries, in the absence of an anterior communicating artery, unite to form a common stem which later divides into two branches is not so rare as might be supposed. Adachi,[4] summarizing the findings of several observers, stated that it was present 17 times in 1,470 brains of Europeans (1.2 per cent). It conforms in general with the primitive mammalian type of distribution of the anterior cerebral arteries.

CASE 2.—*Thrombosis of the basilar artery.*

History.—T. S. aged 42, a machinist, admitted to the medical service, Toronto General Hospital, on Oct. 5, 1931, was well until two days before, when throbbing bitemporal headache developed soon after the evening meal. This continued until he fell asleep. When he awakened at about 2 a. m., the headache was severe; the left arm and leg were numb, and he was unable to move them. At about 4 o'clock he vomited; at this time it was possible to move the arm and leg, but on attempting to walk he had to support himself with the right hand. Improvement continued during the following day so that he could walk without support. On the morning of admission he saw everything double for about thirty minutes, and it was decided to bring him to the hospital.

Twenty years before, the patient had had a penile sore which lasted for about two weeks. Eighteen years before, he was thrown over the handle-bars of a bicycle, striking the left side of the head on the pavement, which rendered him unconscious. He was in bed for one month following the accident with concussion of the brain. During the past year he had suffered from frequent colds in the head and was troubled with postnasal discharge. Three weeks previously, while in the hospital undergoing a minor operation on the heel, he was discovered to have glycosuria. This was being investigated in the medical outpatient department. He had not been subject to headache, shortness of breath or precordial pain.

Examination.—The patient was well nourished and lay in bed. He cooperated well and was emotionally stable. Examination of the cranial nerves revealed nothing abnormal apart from right inferior quadrantic hemianopia and slight dysarthria when test phrases were employed. There was bilateral arcus senilis, and the retinal vessels showed changes characteristic of early arteriosclerosis. The left arm was a little weaker than the right. Tone was slightly diminished in the left arm and leg. Coordination was not as accurate in the left upper limb as in the right. The tendon reflexes were equal and active on the two sides; there was bilateral plantar flexion; there was no clonus. Station and gait were normal, and there was no sphincter disturbance. There was no impairment of any form of sensation. The lungs and abdomen were clear. The radial and brachial arteries were thickened and freely palpable, but not tortuous or beaded. The left temporal artery was markedly thickened. The blood pressure was 120 systolic and 84 diastolic. The pulse was of good quality, and regular in force and rhythm. The heart was not grossly enlarged to percussion; the sounds were of fair quality; there were no murmurs.

The blood showed: hemoglobin, 110 per cent; red cells, 5,500,000; white cells, 7,000; polymorphonuclears, 63 per cent. The Wassermann test was very strongly positive. The urine was normal, apart from the presence of a moderate amount of sugar. The cerebrospinal fluid was under a pressure of 110 mm.; it was clear

4. Adachi, B.: Das Arteriensystem der Japaner, Tokyo, Maruzen Co., 1928, vol. 1.

and showed 6 mononuclears per cubic millimeter; the Pandy test revealed a trace of globulin; the Ross-Jones test was negative; the total protein was 35 mg. per hundred cubic centimeters; the Wassermann test was negative, and the Kahn test 4 +; the colloidal gold curve was negative. Roentgen examination of the skull revealed nothing abnormal. The results of the sugar tolerance test (100 Gm. of dextrose) are recorded in the table:

Results of the Sugar Tolerance Test

Time of Observation	Blood	Urine
Fasting	0.099	0
½ hour	0.158	+ (1.0%)
1 hour	0.156	+++ (2.9%)
2 hours	0.143	+++ (3.0%)
3 hours	0.163	+++ (2.6%)

Dr. W. R. Campbell made the following comment: "The patient has no history of diabetic symptoms; glycosuria has been found on several occasions, and the dextrose tolerance test shows a low threshold for excretion of dextrose and fixation of the curve at about the threshold level. This makes renal glycosuria improbable, while it is not consistent with diabetes mellitus. The condition may be diabetes innocens or possibly an impairment of sugar metabolism associated with syphilis or with the intracranial lesion."

Diagnosis.—The diagnosis was cerebrospinal syphilis, chronic sinusitis and generalized arteriosclerosis.

Medication.—Mercurial inunctions were given daily; potassium iodide, 15 grains (0.9 Gm.), three times a day, and neoarsphenamine 0.45 Gm. intravenously.

Course.—On October 10, the slight weakness, incoordination and hypotonia in the left arm had completely disappeared, and apart from mild headache the patient felt well. The homonymous field defect remained unaltered. On October 18, he awakened with diplopia. He was very emotional and showed slurring dysarthria. There were paresis of the left rectus muscle and coarse horizontal nystagmus on conjugate lateral deviation to both sides. Power in the left leg was impaired at all joints as compared with the right. The tendon reflexes throughout were more active than formerly but were equal on the two sides. There was bilateral dorsiflexion on plantar stimulation, and exhaustible clonus was noted in both ankles. The cerebrospinal fluid was under a pressure of 120 mm. It was clear and showed 20 mononuclears per cubic millimeter; Pandy and Ross-Jones tests, negative; total protein, 35 mg. per hundred cubic centimeters; Wassermann test, 4 +; Kahn test, 4 +; colloidal gold test, negative. Arsphenamine, 0.45 Gm., was given intravenously.

On October 19, the strabismus and diplopia had disappeared. Only a few nystagmoid jerks were elicitable on conjugate deviation of the eyes. The patient was cooperative, but perception was slow. He was emotional and cried frequently. Test phrases were articulated moderately well, but spontaneous speech was slurred and at times unintelligible. There was no paralysis of the tongue or palate. There was marked general weakness of all four limbs. The tendon reflexes were all active. The left plantar response was in flexion; the right was biphasic. There was considerable expectoration of mucus. The respirations were not increased. The chest was clear except for a few coarse râles at the base of the right lung posteriorly.

On October 20, the patient had diplopia on awakening in the morning. A convergent strabismus was present due to paralysis of the left external rectus. Coarse

horizontal nystagmus was elicited on conjugate deviation to the right, and vertical nystagmus on conjugate deviation upward. The pupils were small, the right slightly larger than the left; they reacted well to light and on convergence. A slight left lower facial weakness was present on voluntary movement. The right arm and leg were markedly weak as compared with the left. No alteration in tone was noted. The tendon jerks were active and equal except that the right knee jerk was more easily elicited than the left. The right plantar reflex was in dorsiflexion.

The diagnosis was thrombosis of an artery in the pons. Neoarsphenamine, 0.45 Gm., was given intravenously.

On October 21, the condition was unchanged except that the strabismus was less marked. The left biceps jerk was more active than the right. On October 22, there was dysphagia. The palate moved well in the midline. No alteration in palatal sensation was detected. A gag reflex was present. The vocal cords moved normally. The dysarthria was present as formerly. The tongue protruded well and without deviation. The pulmonary signs previously noted had increased. For the first time there was fever, the temperature being 101 F.

On October 23, the paralysis of the right arm and leg had increased. Both limbs were flaccid, and scarcely any voluntary power remained. The dysphagia became very severe; every attempt to take fluids produced violent spasms of coughing. The patient was rational and well oriented until fifteen minutes before death. At this time he became cyanosed; the respirations were slow, with variable periods of apnea; the temperature was 100; the pulse was imperceptible at the wrist, and the heart sounds were faint and irregular. The patient died while deeply cyanosed.

Final Diagnosis.—The diagnosis was syphilitic endarteritis, with thrombosis of the vessels of the brain stem.

Postmortem Examination.—When the skull was opened, free pus was found in the sphenoid sinuses. Examination of the brain showed a thrombus occluding the left vertebral and the basilar arteries. The other arteries of the circle of Willis and the cerebellar arteries were narrow; their walls were thin and pliable, and an occasional yellow fleck was seen. The internal carotid arteries were almost stenosed by opaque yellow thickenings of their intimal coats. There was no exudate visible, and the ependymal lining of the ventricles was unaltered. The meninges were not adherent, except near the pole of the left temporal lobe where an irregular grayish thickening of the leptomeninges, about 1 cm. in diameter, was adherent to the cortex.

Professor Klotz made the following report on sections of the cerebral arteries: "The sections of the thrombosed basilar artery show a mass of blood clot which is attached to the vessel wall and in places is composed of a structureless granular substance showing evidence of early organization. There are also irregular spaces between the denser masses of clot where a more recent thrombus was disposed. There is no evidence in the section of an old endarteritis. The artery is narrow and distended and shows patches of mucoid degeneration in the middle coat. The elastic membrane is intact throughout the entire circumference save at one point where a dissecting aneurysm extends over about one sixth of the circumference and is filled with recently clotted blood. Its outer wall is made up of the adventitia and outer portions of the medial coat while the inner border is composed of a narrow strip of media and the intima. The muscle tissue of the media at the border of the sac shows degeneration and necrosis without evidence of an inflammatory response. There is nothing in the vicinity of this aneurysm bearing on its

mode of origin save the degenerative changes in the muscle tissue. At some distance from the aneurysmal sac, a small area of necrosis is seen in the outer portion of the media accompanied by a small collection of inflammatory cells. There is no evidence of a syphilitic process. Sections of an anterior cerebral artery show a normal structure. Sections of the middle cerebral artery show a well marked endarteritis producing a nodule of fibrous tissue projecting into the lumen of the vessel and causing some obstruction of it. The arrangement of the connective tissue within the nodule suggests an acute inflammatory process as its mode of origin. The connective tissue is not laid down in laminae but irregularly disposed with some lymphocytes remaining. The media opposite the nodule is unchanged; in the adventitia a collection of lymphocytes is seen. This lymphocytic aggregation has no particular arrangement, nor is necrosis present in the structure. One or two plasma cells are seen but no endothelial cells. In the absence of any further change, I hesitate to suggest a syphilitic background for the inflammatory response, and in support of this I fail to find any reaction in the media. In one specimen the media shows marked atrophy opposite the endarteritic nodule, but this atrophy is unaccompanied by inflammation or necrosis."

Microscopic examination of sections from the occipital lobes, internal capsule and basal ganglia showed no evidence of any inflammatory process. There was increased vascularity, and several corpora amylacea were noted; but there was no cellular infiltration, perivascular or otherwise, and no compound granular corpuscles were seen. A section was taken from the left temporal lobe where the thickened meninges were adherent to the cortex. This was seen to consist of a well marked thickening of the pia-arachnoid so that the normal spaces were almost obliterated. The thickening was the result of an increase in the connective tissue structure, mature in type, without evidence of old blood pigment. Numerous capillaries and arterioles were seen in the deeper portions of the scar tissue and over the surface of the brain in this region. There was a diffuse infiltration with lymphocytes, plasma cells and endothelial cells in the reticular tissue over the surface of the brain, but there was no perivascular mantling of cells. The cortex underlying the scar was compressed and indented and showed reduction in cellular elements. There was an increase in the connective tissue throughout this region.

Apart from the condition in the brain, the other important observation at autopsy was thrombosis of both coronary arteries. The thrombus in the right coronary showed evidence of recanalization, but that in the left was of more recent origin. There was thickening of the intima of both arteries with atheroma and calcification. The media and adventitia were normal apart from some thinning of the media in the regions where the greatest intimal thickening was found. The aorta was thin-walled and elastic throughout and showed a mild degree of chronic nodular endarteritis but no evidence of syphilitic aortitis. Examination of the pancreas, grossly and microscopically, failed to reveal any evidence of pathologic change.

Summary.—A man, aged 42, previously well except for frequent head colds and postnasal discharge, suddenly had severe headache, followed shortly by a left hemiplegia and transitory diplopia. In the hospital, two days later, examination gave negative results except for slight residual signs of hemiplegia and right quadrantic homonymous hemianopia. The Wassermann test of the blood was strongly positive, and the cerebrospinal fluid showed a strongly positive Kahn reaction but was otherwise not abnormal. There was glycosuria which investi-

gation indicated was of intracranial origin. Two weeks after admission, there developed divergent strabismus, dysarthria, nystagmus and weakness of the left leg. These symptoms tended to improve, but three days later convergent strabismus, left facial weakness and right hemiparesis developed. The pupils were very small. Death occurred suddenly from respiratory and cardiac failure. Postmortem examination showed thrombosis of the basilar and left vertebral arteries, advanced cerebral arteriosclerosis and acute sphenoidal sinusitis. No evidence of syphilitic disease was found.

Comment.—The clinical picture in this case is characteristic of a lesion of the brain stem as evidenced by bilateral pyramidal disease affecting the limbs, the cranial nerve palsies, which at times were on the side opposite to the paralyzed limbs, the nystagmus, the pupillary changes, the glycosuria and the termination suggesting medullary failure. The transitory nature of the early paralysis may have been due to spasm of the diseased arteries or to partial occlusion with reestablishment of the circulation by blood from the vessels of the circle of Willis above. The only neurologic finding which could not be explained by the location of the vascular lesion was the right quadrantic homonymous hemianopia, which did not give rise to any symptoms but was discovered during routine examination. There was a history of a severe fall on the left side of the head in 1914, followed by concussion for which he was hospitalized a month. At autopsy there was a small area of scar tissue involving the meninges and brain on the anterolateral surface of the left temporal lobe. It is probable that this was secondary to the cerebral trauma and that it caused the field defect which had been present unnoticed. The thrombosis of the coronary arteries was an interesting finding in association with the cerebral thrombosis. The attacks must have been of the rare painless variety, as the patient gave no history suggesting them, and no suspicion of the presence of the cardiac lesions existed before death. The finding of acute sphenoidal sinusitis post mortem raises the question of the part it played in the development of the cerebral vascular lesion. It is well known that the possibility of thrombosis in atheromatous vessels is increased by the presence of acute infections, and although no direct relationship can be proved in this case, the acute sphenoidal sinusitis cannot be dismissed as a possible etiologic factor. There was no definite evidence of an acute inflammatory origin for the dissecting aneurysm in the basilar artery. The presence of latent syphilis, acquired twenty years previously, is of interest. On admission the Wassermann test of the blood was strongly positive. The cerebrospinal fluid gave a strongly positive Kahn test, but the Wassermann test was negative. The absence of marked increase in the cell count and in the globulin was taken as evidence against a

syphilitic meningeal process being responsible for the symptoms, but it was thought that syphilitic arteritis in the brain stem was a possibility. Following ten days of antisyphilitic treatment, including one injection of neoarsphenamine, the Wassermann and Kahn tests of the cerebrospinal fluid were strongly positive. However, postmortem examination failed to reveal any pathologic condition in the bodily organs, including the brain, that could be attributed to syphilis.

CASE 3.—*Thrombosis of the internal carotid artery.*

History.—C. S., a laborer, aged 58, admitted to the medical service of the Toronto General Hospital on Feb. 27, 1932, with paralysis of the left arm and leg of six hours' duration, had been known to have diabetes for more than a year, but the disease had been controlled by diet alone. Two weeks before his admission to the hospital he contracted pneumonia. The "crisis" occurred on the ninth day, and he was thought to be progressing favorably. On the evening of admission he was observed suddenly to turn a queer color and shortly afterward to be unable to speak or to move the left arm or leg. His previous health had been good, and there was nothing important in the family or personal history.

Examination.—The patient was semicomatose, mumbled inarticulately and was entirely uncooperative. The fundi were regarded as normal at this time. There was conjugate deviation of the eyes to the right with slight ptosis of the right upper eyelid. The pupils were equal in size; the left pupil reacted briskly to light; the right pupil gave no direct reaction to light but reacted consensually. Sensation to pin prick was impaired over the left side of the face, and the left corneal reflex was sluggish. A left lower facial weakness was evident, the mouth being drawn to the right. The remaining cranial nerves could not be tested owing to lack of cooperation. There was flaccid paralysis of the left arm and leg, involving the arm more than the leg. The tendon reflexes were more active in the left arm and leg than in the right. The left plantar reflex was in dorsiflexion. Pin prick was not appreciated over the left arm; it was appreciated equally well in the two legs. There was moderate thickening of the radial and brachial arteries. The pulse was regular in force and rhythm; the rate was 57 per minute. The heart was not grossly enlarged; the sounds were poorly heard but of fair quality; there were no murmurs. The blood pressure was 125 systolic and 80 diastolic, it rose to 160 systolic and 90 diastolic two hours after admission. Respirations were stertorous, 24 per minute. Examination of the chest showed evidence of resolving bronchopneumonia at the base of the left lung. Examination of the abdomen gave negative results.

The urine showed: specific gravity, 1.028; some sugar; acetone, a trace; albumin, none. The blood showed: hemoglobin, 98 per cent; red cells, 5,200,000; white cells, 24,000; blood sugar during fasting, 0.166; Wassermann test, negative.

Course.—On March 3, the temperature, which was subnormal on admission, had risen to 102.4 F. An increase in the moisture at the base of the left lung was found. The patient was delirious, but articulated well. He would cry out to imaginary people and converse with them, then periodically lapse into a comatose state of varying duration. The left optic disk was normal in appearance. The right optic disk and the surrounding retina were very pale; there was a moderate amount of edema about the disk and covering it. The arteries were very small and in places not visible. Many of them presented a beaded appearance. Between these pieces of clot the thin shell-like outline of the vessel walls could be seen. There was no cherry-red appearance of the macula. Conjugate deviation of the

eyes to the right was present, and they could not readily be made to move to the left. On rotating the head the eyes would swing toward the midline, revealing a divergent strabismus due to a lagging of the right eye. It was ascertained that the right eye was totally blind. A temporal field defect was demonstrated in the left eye. The pupils were of moderate size and approximately equal. The right pupil gave no direct reaction to light. The left hemiplegia had altered since admission. The facial weakness, while still evident, was less apparent, and there was marked improvement in the power of the left leg. No spontaneous movement of the limb was noted, but on stimulation with a pin a fairly strong flexion withdrawal occurred. The paralysis of the left arm was unchanged. The reflexes were as previously noted. Sensation to pin prick was still impaired over the left side of the face and left arm but was good on the left leg. The cerebrospinal fluid was under a positive pressure of 165 mm.; it was clear and showed 32 cells, 70 per cent mononuclears. The Pandy test was positive.

During the following week the patient gradually lapsed into deep coma. His temperature continued to rise, and the signs of pulmonary involvement increased. He died on March 10.

Diagnosis.—The diagnosis was thrombosis of the right internal carotid artery.

Postmortem Examination.—When the brain was removed, the right cerebral hemisphere appeared larger than the left and was soft on palpation. The right internal carotid artery, as it passed through the carotid canal, was occluded by a soft, dark red thrombus. The thrombus ended at the point where the internal carotid divided into its two terminal branches. There was also a thrombus blocking the right middle cerebral artery as it lay in the sylvian fissure. The vessels of the circle of Willis showed occasional yellowish thickenings in their walls but were not markedly altered in appearance. After fixation, a coronal section through both hemispheres anterior to the tips of the temporal lobes showed a large area of softening, mostly distributed in the region supplied by the right middle cerebral artery. Petechial hemorrhages were present in large numbers throughout the cortex in the softened area. A second coronal section immediately behind the optic chiasm showed extensive softening, again in the distribution of the right middle cerebral artery, with numerous petechial hemorrhages in the subcortical white matter. A third coronal section passing through the midbrain showed petechial hemorrhages in the cortex, but no definite evidence of softening in the white matter. In these sections the area of distribution of the anterior cerebral artery showed little or no softening. Sections of the thrombosed internal carotid artery were examined by Professor Klotz, who reported: "The portion of the internal carotid artery which was cut showed relatively little change in its wall. The intima was slightly thickened, but not to an extent to encroach on the lumen. The internal elastic tissue showed splitting of its layers and occasional areas of calcification. The media appeared intact without the presence of inflammatory cells or new vasa vasorum. There was some lymphocytic infiltration in the adventitia and in the perivascular structures. The lumen of the vessel was occupied by a recent laminate thrombus which at some points of its attachment showed the earliest beginning of cellular proliferation from the intima. It is probable that the sections did not pass through the point of origin of the thrombus. There was neither extensive sclerosis of the intima nor inflammatory reaction of the vessel wall to suggest the reason for the development of the thrombus."

Sections of the right middle cerebral artery showed, likewise, very little change in its wall. The lumen contained a thrombus in the early stages of organization. Sections of the right anterior cerebral artery revealed no endarteritis. A small

mural thrombus was present. A section through the right optic nerve immediately behind the eyeball showed evidence of early demyelinization. In this section the central artery of the retina contained no thrombus, but there was a large amount of serum present in its lumen. Sections of the cortex and white matter from the right precentral gyrus (cortical leg area) showed marked chromatolysis of the Betz cells and early demyelinization of the white matter. Similar sections from the left hemisphere revealed a normal structure.

Apart from the changes in the brain, other observations made post mortem included: recent thrombosis of both femoral veins, bilateral pulmonary embolism and bilateral bronchopneumonia in the lower lobes. The heart and aorta were essentially normal in appearance.

Summary.—A man, aged 58, who had been undergoing dietary treatment for a mild diabetic condition, while convalescing from pneumonia suddenly had paralysis of the left arm and leg. Six hours later, he was semicomatose; the right pupil failed to react to light; severe left hemiplegia was present, involving the face, arm and leg, and sensory impairment over the paralyzed area in the face and left arm. Examination on the third day after admission showed blindness in the right eye; ophthalmoscopic examination indicated that thrombosis of the central artery of the retina was the cause. The left hemiplegia had improved, particularly in the leg. During the following week he became increasingly comatose, and signs of extensive pulmonary involvement appeared. He died on the tenth day after the onset of the hemiplegia. Post mortem the right internal carotid artery was occluded by a thrombus and, likewise, the right middle cerebral artery as it lay in the sylvian fissure. There was a large area of softening in the distribution of the right middle cerebral artery. The cerebral arteries showed only moderate degenerative changes in their walls.

Comment.—The syndrome known as "carotid hemiplegia" is well illustrated by this case. It is recognized by blindness of the eye on the side of the lesion and hemiplegia on the opposite side. The ophthalmic artery arises from the internal carotid just as the latter emerges from the cavernous sinus. Accordingly, occlusion of the internal carotid artery below the point where this branch comes off results in ischemia of the optic nerve. The hemiplegia is produced by ischemia of the brain in the distribution of the terminal branches of the internal carotid artery—the anterior and middle cerebral arteries.

Carotid hemiplegia is more frequently the result of embolism than of primary thrombosis. When embolism is the cause, complete recovery from the hemiplegia often occurs as a result of reestablishment of circulation through the circle of Willis. The blindness remains, however, owing to secondary thrombosis extending through the ophthalmic artery (Collier).[5]

5. Collier, J., in Price, F. W.: A Textbook of the Practice of Medicine, ed. 3, New York, Oxford University Press, 1930, p. 1447.

The partial recovery from the paralysis which occurred in this case during the three days following the onset was due, no doubt, to the reestablishment of circulation through the circle of Willis. It is probable that during this period the reestablished circulation was fairly adequate through the right anterior cerebral artery by way of the left anterior cerebral and anterior communicating arteries, whereas the sylvian branch of the right middle cerebral, found post mortem to be thrombosed, was conveying a partial and inadequate blood supply. In this way the improvement, which was maximal in the leg, can be accounted for. Post mortem, the right paracentral lobule (cortical leg area) showed degenerative changes microscopically, indicating that the distribution of the anterior cerebral artery suffered a partial ischemia, though not so severe as that in the middle cerebral artery which gave rise to gross softening.

The internal carotid artery is not commonly the site of primary thrombosis, and the reason for the occurrence of a thrombus in it in this case is not clear. The thrombosed artery and the other cerebral arteries examined showed less atheromatous change than might be expected considering the patient's age.

It is probable that the pneumonia was a predisposing factor in the development of the thrombosis. The fact that thrombi were present in both femoral veins suggests the possibility that the infection produced a general tendency to thrombosis.

SUMMARY

The clinical histories and pathologic observations in three cases of cerebral thrombosis have been described, illustrating the syndromes associated with softening in the distribution of the anterior cerebral, basilar and internal carotid arteries respectively. An attempt has been made in each case to correlate the clinical symptoms with the pathologic observations.

THE ARGYLL ROBERTSON PUPIL

AN ANATOMIC-PHYSIOLOGIC EXPLANATION OF THE PHENOMENON, WITH A SURVEY OF ITS OCCURRENCE IN NEUROSYPHILIS

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In 1869, Argyll Robertson, in an article [1] entitled, "Four Cases of Spinal Myosis, with Remarks on the Action of Light on the Pupils," described a peculiar pupillary phenomenon. At first this phenomenon was believed to have a pathognomonic significance, but more recently it has lost some of its diagnostic importance. This can probably be explained by the following facts: (1) The anatomic and physiologic bases for this unusual pupillary disturbance have been imperfectly understood, and (2) confusion has developed as to the exact definition of the phenomenon. In the last decade there have been some excellent anatomic and physiologic studies on the pathways of the light reflex and also on the pathways for the pupillodilator fibers. These separate studies, when put together, give for the first time an exact anatomic location for a lesion which would explain all the phenomena in the Argyll Robertson pupil.

It is our purpose in this article, therefore, to bring these anatomic and physiologic studies together and to show how they explain the abnormality of the pupil described by Argyll Robertson. It is also our purpose to call attention to the original definition and to determine if an adherence to it would increase the clinical value of the sign. In addition, we have sought to determine the relative frequency of this sign in the various types of neurosyphilis by an analysis of the records in 749 cases of neurosyphilis that have recently come under observation at the Boston City Hospital and the Boston Psychopathic Hospital.

DEFINITION OF THE ARGYLL ROBERTSON PUPIL

In his original description of four cases Robertson [1] stated:

> Although the retina is quite sensitive and the pupil contracts during the act of accommodation for near objects, yet an alteration in the amount of light admitted

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1. Argyll Robertson, D.: On an Interesting Series of Eye Symptoms in a Case of Spinal Disease, with Remarks on the Action of Belladonna on the Iris, Edinburgh M. J. **14**:696, 1869; Four Cases of Spinal Myosis, with Remarks on the Action of Light on the Pupil, ibid. **15**:487, 1869.

to the eye does not influence the size of the pupil. This can not be explained by the supposition that the pupil is already so small as to be incapable of further contraction under light, because in the healthy eye a still further degree of contraction may be effected by the use of the Calabar bean, and yet the pupil varies in size according to the intensity of the light.

He states further:

These four cases serve well to illustrate the connection between certain eye symptoms and a diseased condition of the spinal cord. In all of them there was marked contraction of the pupil which differed from myosis due to other causes, in that the pupil was insensible to light, but contracted still further during the act of accommodation for near objects, while strong solutions of atropine only induced a medium dilatation of the pupil.

From these statements it can be seen that the abnormality described by Robertson has five characteristics: (1) The retina is quite sensitive. (2) The pupils are small (miotic). (3) They remain of a constant size regardless of the intensity of the illumination admitted to the eyes. (4) They contract actively on accommodation for near objects. (5) They dilate imperfectly on instillations of atropine.

Since this original description, one additional feature has been found to be constantly present in Argyll Robertson pupils, that is: (6) The absence of dilatation of the pupils when painful stimuli are applied to the skin.

In attempting to analyze these characteristics of the Argyll Robertson pupil with regard to their pathologic and physiologic significance, the following interpretations may be made:

1. "The retina is quite sensitive": This implies that the retina and therefore the optic visual pathways are relatively intact.

2. "The pupils are small": Small pupils (miosis) can be due only to stimulation of the parasympathetic or paralysis of the sympathetic control of the eye. Since the miosis persists for years, it is unlikely that it is due to parasympathetic stimulation.

3. "The pupils remain of a constant size regardless of the intensity of the illumination admitted to the eyes": This can be explained only by a paralysis of the light reflex fibers or a paralysis of the innervation of the iris. That the latter is not the cause is shown by the next characteristic.

4. "They contract actively on accommodation for near objects": This implies that the cortical pathway for accommodation, the third nerve nucleus and the entire pathway from the nucleus to the ciliary muscle are intact.

5. "They dilate imperfectly in response to instillations of atropine": This is characteristic of paralysis of the sympathetic innervation of the eye.

6. "There is no dilatation of the pupils in response to painful stimuli": The work of Karplus and Kreidl [2] proved that the dilatation of the pupil on painful stimulation is mediated through the sympathetic center in the hypothalamus, and that it disappears when this center is extirpated. Removal of the cerebral hemispheres has no effect on this reaction, provided the hypothalamic center remains intact.

From this it is apparent that the phenomena found in the Argyll Robertson pupil can be produced only by an injury to both the light reflex and the sympathetic fibers. Also, if the phenomena are to be explained on the basis of a single lesion, it must be at a point where the two pathways converge. It is also evident from the foregoing analysis that in view of the preservation of certain normal characteristics (such as sensitive retina and normal accommodation reflex) it is unlikely that this point can be in the optic nerve, the third nerve nucleus or its peripheral connections.

Recent anatomic studies on the pupillomotor and the sympathetic pathways to the eye have shown that these two pathways intermingle in the tegmentum just ventrally to the posterior commissure where their fibers run together for a short distance.

ANATOMY AND PHYSIOLOGY OF THE PUPILLARY REACTIONS

In the light of recent investigations, present knowledge as to the anatomic pathways concerned in the pupillary reactions may be summarized as follows:

(*a*) *Pupillomotor Fibers.*—Karplus and Kreidl [3] showed by stimulation and extirpation experiments in apes, cats and dogs that the afferent fibers for the pupillary light reflex originate in the retina. These fibers decussate in the optic chiasm in the same manner as the visual fibers. Stimulation of the optic tract between the chiasm and the lateral geniculate body caused a constriction of the pupils. The light reflex fibers do not enter the lateral geniculate body, but pass with the brachium of the superior corpora quadrigemina toward this body. Stimulation of the brachium caused constriction of the pupils. Karplus and Kreidl were unable to trace the light reflex fibers further, as stimulation of the anterior colliculus always caused dilatation of the pupils. With the Bielschowsky silver impregnation method, Lenz [4] showed that fibers from the optic nerve pass with the brachium of the superior colliculus to the cephalad end of the superior colliculus. A portion of

2. Karplus, J. P., and Kreidl, A.: Gehirn und Sympathicus: II. Mitteilung. Ein Sympathicuszentrum im Zwischenhirn, Arch. f. d. ges. Physiol. **135**:401, 1910.

3. Karplus, J. P., and Kreidl, A.: Ueber experimentelle reflektorische Pupillenstarre, Neurol. Zentralbl. **32**:82, 1913.

4. Lenz, G.: Untersuchungen über die intrazerebrale Bahn des Pupillarreflexes: Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch. **46**:140, 1927.

the fibers cross in the dorsal portion of the posterior commissure, while the remainder arch ventrally toward the oculomotor nuclei. From the stimulation experiments of Karplus and Kreidl it was assumed that these were the light reflex fibers, but it remained for Ranson [5] to prove this assumption. Using the Horsley-Clark apparatus, Ranson stimulated the various parts of the pupillary constrictor pathway. When the superior quadrigeminal brachium was stimulated, constriction of the pupils was obtained; constriction also occurred when the posterior commissure was stimulated. Except for a small tract in the pretectal region between the posterior commissure and the oculomotor nucleus, where stimulation produced constriction of the homolateral pupil only, stimulation in the region of the anterior colliculus produced dilatation of the pupils. Ranson's work shows that the light reflex fibers, which cross in the optic chiasm, recross in the posterior commissure. This is of great importance, because a destruction of the light reflex pathway after these fibers have recrossed would cause unilateral Argyll Robertson pupil. No theories as to the localization of the lesion producing the phenomenon of Argyll Robertson pupil are valid unless they also explain the unilateral occurrence. The pathway of the efferent pupillomotor fibers from the oculomotor nucleus is by way of the oculomotor nerve to the ciliary ganglion and thence via short ciliary nerves to the ciliary muscle. There is one other point of considerable importance with regard to the pupillomotor fibers, and that is their anatomic independence of the visual fibers. Von Monakow [6] showed that in the optic nerve there are two types of fibers, thick and thin, and that only the thick fibers go to the anterior colliculus. Also the clinical observations [7] in cases of injury to or inflammation of the optic nerve, when there was a loss of the light reflex with little or no impairment of the vision, indicate that these pathways are physiologically separate, and that they may have a separate vulnerability.

(*b*) *Pupillodilator Fibers.*—Karplus and Kriedl,[8] in their experiments, found a sharply localized area in the hypothalamus stimulation of which produced dilatation of the pupil. They did not, however, trace the course of these fibers from this area to the midbrain, although they did find that stimulation of the anterior corpora quadrigemina

5. Ranson, S. W.: The Pathway of the Pupillary Light Reflex; read before the Fifty-Ninth Annual Meeting of the American Neurological Association, Washington, D. C., May 9, 1933.

6. von Monakow: Gehirnpathologie, Vienna, Alfred Hölder, 1905.

7. Wilson, S. A. K.: Some Problems in Neurology: The Argyll Robertson Pupil, J. Neurol. & Psychopath. **2**:1, 1921.

8. Karplus, J. P., and Kreidl, A.: Gehirn und Sympathicus: I. Zwischenhirnbasis und Halssympathicus, Arch. f. d. ges. Physiol. **129**:138, 1909; II. Ein Sympathicuszentrum im Zwischenhirn, ibid. **135**:401, 1910; III. Sympathicusleitung im Gehirn und Halsmark, ibid. **143**:109, 1911-1912.

produced dilatation of the pupil. This, however, was recently investigated further by Beattie [9] and his co-workers. They localized a center in the posterior portion of the hypothalamus which controls the true sympathetic nervous system. They showed by the degeneration preparations of Marchi and Alzheimer-Mann, after experimental extirpation of this center, that fibers running from it enter the anterior colliculus by passing ventrally to the posterior commissure. From there a portion of the fibers arch ventrally to enter the mediolateral portion of the midbrain and descend in the reticular formation of the pons and medulla. Another portion arches ventromedially to run in the homolateral and contralateral posterior longitudinal bundle.[10] They found that the pupillodilator fibers were uncrossed and were in connection only with the homolateral cervical sympathetic center. Thus it can be seen that in passing ventrally they are in close connection with the pupillomotor fibers, and it was due to stimulation of these sympathetic fibers that Karplus and Kreidl [2] always noted a dilatation of the pupil instead of the expected contraction when they stimulated the anterior colliculus. The further course of the pupillodilator fibers caudad through the medulla is well known from the clinicopathologic observations in cases of thrombosis of the posterior inferior cerebellar artery.[11] These fibers end in the sympathetic cells in the lower cervical segments of the cord. The impulses then pass by way of the superior cervical sympathetic ganglion to the ciliary ganglion and thence by the short ciliary nerves to the dilating muscles of the iris.

(*c*) *Pathway of the Accommodation Reflex.*—Since accommodation is in the broader sense a voluntary movement, the origin is supposed to be in the cerebral cortex. From there fibers pass by the internal capsule and the cerebral peduncles to the oculomotor nucleus. The peripheral pathway is exactly similar to that of the pupillomotor fibers.

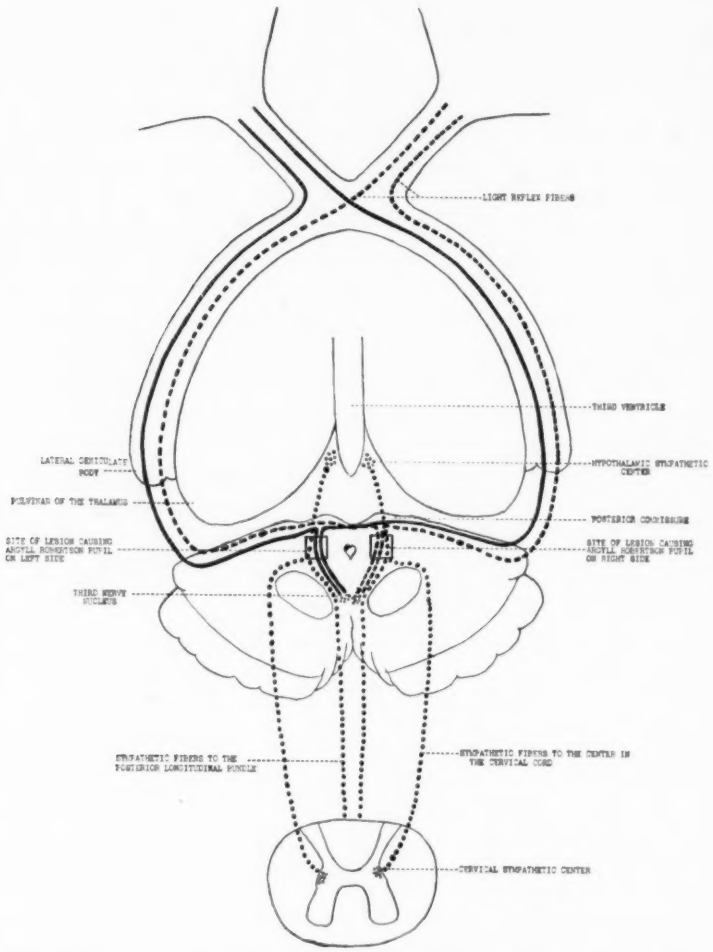
Comment.—From the facts outlined, it can be seen that a single lesion can cause all the phenomena of the Argyll Robertson pupil. This lesion would have to be in the tegmentum ventrally to the posterior

9. Beattie, J.; Brow, G. R., and Long, C. N. H.: Physiological and Anatomical Evidence for the Existence of Nerve Tracts Connecting the Hypothalamus with Spinal Sympathetic Centers, Proc. Roy. Soc., London, s.B, **106**:253, 1930. Beattie, J.; Duel, A., and Ballance, C.: The Effects of Stimulation of the Hypothalamic Pupillo-Dilator Centre after Successful Anastomoses Between the Cervical Sympathetic and Certain Motor Nerves, J. Anat. **66**:283, 1932.

10. The destruction of these fibers to the posterior longitudinal bundle explains the absence of pupillary reaction to vestibular stimulation which is often found in cases showing Argyll Robertson phenomena (Spiegel, E. A.: Zur Lokalisation des Argyll Robertsonschen Phänomens [insbesondere nach Verhalten der vestibulären Pupillenreaktion], Wien. klin. Wchnschr. **38**:189, 1925).

11. Merritt, H. H., and Finland, M.: Vascular Lesions of the Hind-Brain (Lateral Medullary Syndrome), Brain **53**:290, 1930.

commissure, where the light reflex fibers are coursing ventromedially to the oculomotor nucleus and where the sympathetic fibers are also coursing ventromedially to the posterior longitudinal bundle and ventro-


LIGHT REFLEX FIBERS
THIRD VENTRICLE
LATERAL GENICULATE BODY
HYPOTHALAMIC SYMPATHETIC CENTER
PULVINAR OF THE THALAMUS
POSTERIOR COMMISSURE
SITE OF LESION CAUSING ARGYLL ROBERTSON PUPIL ON LEFT SIDE
SITE OF LESION CAUSING ARGYLL ROBERTSON PUPIL ON RIGHT SIDE
THIRD NERVE NUCLEUS
SYMPATHETIC FIBERS TO THE POSTERIOR LONGITUDINAL FUNICLE
SYMPATHETIC FIBERS TO THE CENTER IN THE CERVICAL CORD
CERVICAL SYMPATHETIC CENTER

Schematic representation of the pathway of the pupillary light reflex fibers from the retina to the third nerve nucleus, and of the sympathetic pathways from the hypothalamus to the spinal cord. The light reflex fibers from the nasal halves of the retina, which cross in the optic chasm, recross in the posterior commissure. A small lesion just ventrally to the posterior commissure would destroy both the light reflex and sympathetic pathways and produce a homolateral Argyll Robertson pupil.

laterally to the reticular formation. There would be a loss of the light reflex owing to involvement of the light reflex fibers, and a miotic pupil

which would not react to painful stimuli and which would dilate imperfectly to atropine solutions, owing to involvement of the descending pupillodilator fibers. The accommodation reflex would be preserved, since the pathway for accommodation probably enters the oculomotor nucleus ventrally via the cerebral peduncles.

It is believed that this explanation of the production of the Argyll Robertson pupil rests on a firm anatomic foundation and explains the phenomena better than any other heretofore offered.[12] It also explains the occasional unilateral appearance of the phenomenon. Wilson,[7] in 1921, stated that the question as to the location of the lesion causing the Argyll Robertson pupil resolves itself into the location of the point where the light reflex arc and the pupillodilator path come sufficiently close to each other to be simultaneously involved in a common destructive lesion. At that time, however, the pathway of the pupillodilator fibers was not well understood, and Wilson was not able to explain all of the phenomena of the Argyll Robertson pupil on the basis of a single lesion. He did, however, state that everything seemed to point to the anterior portion of the midbrain near the aqueduct of Sylvius as being the site of the lesion.

The studies of Beattie[9] and his co-workers have given the location of the pupillodilator pathways, and since they run close to the light reflex fibers, they give the answer to the question propounded by Wilson.

PATHOGENESIS OF THE ARGYLL ROBERTSON PUPIL

It is easy to understand how an infiltrative lesion, such as a glioma, in the anterior colliculus could destroy the light reflex and the sympathetic fibers and produce the Argyll Robertson pupil, but there has been, as yet, no satisfactory explanation of the cause of this phenomenon in syphilis of the central nervous system.

Several theories have been advanced but none has any conclusive pathologic support. Ingvar[12] explained the reflex immobility of the pupil to light as due to an involvement of the light reflex fibers by chronic meningitis. He claimed that the reflex fibers run along the superficial portion of the optic tract and the anterior colliculus, and that these fibers are easily destroyed by chronic meningitis. Such a lesion could produce a reflex immobility of the pupils and is perhaps the cause of a reflex immobility to light associated with a large pupil, but it would not cause miosis and the other sympathetic phenomena which are part of the Argyll Robertson pupil; also, clinically, it is a well observed fact that the Argyll Robertson pupil is uncommon in the uncomplicated meningeal forms of neurosyphilis. Wilson,[7] who considered that the

12. Ingvar, S.: On the Pathogenesis of the Argyll Robertson Phenomenon, Bull. Johns Hopkins Hosp. **43**:363, 1928. Wilson.[7]

lesion must be centrally placed in the anterior colliculus, postulated that the degeneration of the fibers might be due to ependymitis. This is a more reasonable explanation, as it is conceivable that both the light reflex and the sympathetic fibers could thus be involved. This is especially true for cases of dementia paralytica, in which ependymitis is practically always found. On the other hand, the Argyll Robertson pupil is just as common in tabes dorsalis, in which there is rarely any pathologic evidence of ependymitis.

It is also possible that the Argyll Robertson pupil may be due to a selective involvement of the light reflex and sympathetic fibers as a result of an especial vulnerability of these fibers to the syphilitic infection, as advocated by Spielmeyer [13] for the dorsal column changes in tabes. But since the light reflex and sympathetic fibers run together in the midbrain, it is more likely that a localized lesion is the cause of the Argyll Robertson pupil. There have been no satisfactory pathologic examinations of this region with silver impregnation methods, and since these fibers are unmyelinated, their degeneration would not be seen in the ordinary myelin sheath preparations.

CLINICAL SIGNIFICANCE OF THE ARGYLL ROBERTSON PUPIL

The confusion in regard to the diagnostic value of the Argyll Robertson pupil is caused, first, by faulty technic in observing and recording the pupillary reactions and, second, by the criteria used in making the diagnosis of this phenomenon. For these reasons it is necessary to emphasize the importance of using a uniform method for testing the reflex and recording the observations. Many records merely contain the statement: "Patient has Argyll Robertson pupil," without any objective description of the size of the pupil, the methods and conditions of testing, or the pupillary responses to these tests. One should be objective and describe accurately the tests and the results obtained. The visual acuity of the patient should be recorded; the size of the pupils should be stated in millimeters, and their contour and shape should be described. It should be noted whether or not they alter in size at any time during the observation, particularly when there are changes in the intensity of the illumination, as when the patient goes into and out of a dark room or when he gazes out of a window. The simplest and most satisfactory method of testing the light reactions of the pupils is to place the patient in front of a bright window, asking him to focus on some distant object, and alternately shade the eye from and expose it to the light, noting any change in the size of the pupil being tested or in the opposite pupil. If a flashlight is used, one must

13. Spielmeyer, W.: Zur Pathogenese der Tabes, Ztschr. f. d. ges. Neurol. u. Psychiat. **84**:257, 1923.

remember that it is a much weaker stimulus than daylight. Care must be taken that the patient does not focus on the light, because the resulting reflex accommodation may be mistaken for a reaction to the light. This can be prevented by throwing the light in from the side of the eye. The accommodation reflex should be tested by having the patient focus alternately on distant and very near objects. The response of the pupil to painful stimuli may be obtained by pinching the neck. The testing of the pupillary response to instillation of atropine solution can be carried out if considered necessary, but if the aforementioned conditions are fulfilled, one can usually determine whether the pupil being tested is abnormal.

If one considers that an Argyll Robertson pupil is any pupil that reacts on accommodation but does not react to light, then it is correct to claim that the Argyll Robertson pupil is not pathognomonic of neurosyphilis. There are many conditions in which there is an absence of pupillary reaction to light but retention of an active accommodation reflex, chief among which is that of a blind eye. In many such cases the patients have been described as presenting Argyll Robertson pupils. This same statement applies to cases of injury to the optic nerve, cases of tumor of the brain in which there may be a choked disk, and cases of multiple sclerosis, alcoholism and other conditions in which there is optic neuritis. In all these there is some degree of visual loss, and since it is known that the vulnerability of the light fibers is different from that of the pupillomotor fibers, one cannot tell from the visual acuity how severe the damage to the pupillomotor fibers is. In most of such cases there is also shown a dilated pupil. If, however, one accepts the criteria as laid down by Robertson in his original observations (which seems only fair if his name is to be attached to the sign), one will see that all amblyopic cases are excluded, as they fail to fulfil one or two of the cardinal requirements. If there is any degree of visual loss due to optic atrophy or optic neuritis, even though the patient has syphilis of the central nervous system, it is unsafe to diagnose the condition as Argyll Robertson pupil, as one cannot estimate how severe the damage to the pupillomotor fibers in the nerve is. If the pupil is of normal size (or larger), if it varies in size from time to time or if it responds to painful stimuli, the diagnosis of an Argyll Robertson pupil is excluded, since evidence of involvement of the pupillodilator fibers is lacking. The only incontrovertible cases of Argyll Robertson pupil occurring in diseases other than neurosyphilis are those due to a gliomatous invasion of the midbrain. This was first emphasized by Wilson.[7] In his case 5, vision was relatively intact; the pupils were miotic and reacted readily on accommodation. They did not react to light or to painful stimuli. Postmortem examination showed a destructive invasion of both anterior colliculi and the peri-aqueductal gray

matter. The description of other cases in the literature are such as to make it impossible to decide whether there was an Argyll Robertson pupil, or whether the loss of the light reflex was due to a concomitant visual loss. Wilson [7] summarized the literature in regard to the non-syphilitic Argyll Robertson pupil, and a review of the cases that he cites and other [14] cases indicates either that there was an associated visual loss (trauma to the optic nerve, optic neuritis, choked disk, amaurosis or other condition), or that the pupils did not show evidences of sympathetic paralysis. Dilated pupils occur in syphilis of the central nervous system, and there is also a certain percentage of cases of neurosyphilis in which visual loss is due to optic neuritis or to primary optic atrophy. Many of these cases show a dissociation in the pupillary reaction, but the pathology of the phenomena in these cases is usually different from that of the Argyll Robertson phenomena. In such cases a lesion can usually be demonstrated in the optic nerve proper or in the third nerve nucleus. The occasional cases of syphilis of the central nervous system in which all the phenomena of the Argyll Robertson pupil are shown except constriction on accommodation are probably due to a more extensive lesion than that occurring in the ordinary case; or there may be concomitant lesions in other locations, e. g., in the third nerve nucleus or the ciliary ganglion.

Many of the older clinicians, such as Bumke [15] and Weiler,[16] insisted that the Argyll Robertson pupil occurs only in syphilis of the central nervous system, and more recently Adie [17] stated that he had never personally seen or found reported in the literature any case satisfying the criteria given by Argyll Robertson in which syphilis of the central nervous system was excluded. He examined a large number of cases of chronic encephalitis, in which pupillary abnormalities are frequent, and never found an Argyll Robertson pupil. Any destructive lesion of the light reflex and the sympathetic fibers in the tegmentum just ventrally to the posterior commissure would produce an Argyll Robertson pupil, but clinical experience has shown that such lesions are so extraordinarily rare, except in neurosyphilis, that for all practical purposes the statement of Adie [17] is true: "The true Argyll Robertson

14. Ford, F. R.: Cholesteatoma of the Third Ventricle, with Bilateral Argyll Robertson Pupil, J. A. M. A. **82**:1046 (March 29) 1924. Moore, R. F.: The Non-Luetic Argyll Robertson Pupil, Tr. Ophth. Soc. U. Kingdom **51**:203, 1931. Moore, R. F.: Non-Luetic Argyll Robertson Pupil, Brit. M. J. **2**:843, 1925. Inman, W. S.: The Non-Luetic Argyll Robertson Pupil, ibid. **2**:1179, 1925.

15. Bumke, O.: Pupillenstörungen bei Geistes und Nervenkrankheiten: Physiologie und Pathologie der Irisbewegungen, Jena, Gustav Fischer, 1911.

16. Weiler, K.: Untersuchungen der Pupille und der Irisbewegungen beim Menschen, Berlin, Julius Springer, 1910.

17. Adie, W. G.: Argyll Robertson Pupils, True and False, Brit. M. J. **2**:136, 1931.

pupil . . . is as near as may be, in an imperfect world, an infallible sign of syphilis of the nervous system."

OCCURRENCE OF THE ARGYLL ROBERTSON PUPIL IN NEUROSYPHILIS

In order to determine the relative frequency of the Argyll Robertson sign in neurosyphilis, we have analyzed the records in 749 cases of neurosyphilis which have come under observation in recent years at the Boston City Hospital and the Boston Psychopathic Hospital. Many of the patients have been examined by us personally. The results are shown in table 1. Considerable difficulty was encountered in classifying the cases, because in many instances there was no objective description of the pupils. The records in many cases merely stated: "The patient has Argyll Robertson pupils," "fixed pupils," "sluggish pupils," and the like. Sufficient description was present, however, to enable us to determine that the true Argyll Robertson pupil was present in 61 cases (8.1 per cent). In 170 cases (22.7 per cent) the record stated that the Argyll Robertson pupil was present, but did not give any objective description; this makes a total of 231 cases (30.8 per cent). In addition, in 56 cases (7.4 per cent) the patients showed incomplete Argyll Robertson pupils. The term incomplete Argyll Robertson pupil is used to mean one which satisfies all the criteria except that the light reaction is only partially lost. Since the Argyll Robertson pupil does not develop suddenly, there will be various stages in its development, and the partial phenomenon probably has a clinical significance similar to the complete phenomenon. Pupillary abnormalities of other types were found in 344 cases (46 per cent), but since such abnormalities occur in numerous conditions, they do not have any specific diagnostic value. Thus in 624 cases (84 per cent) abnormal pupils were found. It is interesting to note that the Argyll Robertson pupil is not always bilateral. In 11 cases (1.4 per cent) it was found that one pupil showed the Argyll Robertson sign, whereas the other pupil showed only minor changes. Table 1 shows the relative incidence of the Argyll Robertson pupils in the various forms of neurosyphilis.

Dementia Paralytica.—In the 208 cases of dementia paralytica reviewed by us the findings were as given in table 2.

These records represent the findings on the first examination, and since the cases were not divided into early and late groups one cannot state how many of these cases would have shown the Argyll Robertson phenomena or abnormal pupils before death, but it is natural to assume that the percentages increased with the progress of the disease.

Probably the largest series of cases of dementia paralytica in which the pupillary reactions have been studied is that reported by Bumke.[15] He collected from the literature and from personal observation 5,020 cases. The percentage of cases in which an absence of the reaction to

light was shown varied between 41.2 and 62, with an average of 45.5. The percentage of cases in which a good reaction to light was found varied between 1.7 and 36, with an average of 26. Bowman and Raymond [18] found normal pupillary reactions in only 8 per cent of 412 cases of dementia paralytica. It is probable, therefore, that the Argyll Robertson pupil is present in about from 40 to 60 per cent (according to the stage of the disease) of all the cases of dementia paralytica, and that absolutely normal pupils are present in less than 10 per cent.

TABLE 1.—*Incidence of Argyll Robertson Pupils in Various Forms of Neurosyphilis (749 Cases)*

	True Argyll Robertson		So-Called Argyll Robertson		Partial Argyll Robertson		Other Abnormalities		Normal		Total
	Number	Per Cent	Number	Per Cent	Number	Per Cent	Number	Per Cent	Number	Per Cent	Number
Dementia paralytica	18	8.7	51	24.6	35	16.9	93	44.3	11	5.4	208
Tabes dorsalis	31	16.7	73	39.2	9	4.9	68	36.4	5	2.6	186
Tabetic form of dementia paralytica	4	12.5	16	50.0	3	9.3	8	24.9	1	3.1	32
Cerebral vascular syphilis	2	3.0	1	1.5	1	1.5	35	53.0	27	40.8	66
Meningovascular syphilis	2	1.1	27	15.1	7	4.0	95	53.0	48	26.7	179
Syphilis of the spinal cord	0	0.0	0	0.0	0	0.0	13	76.4	4	23.5	17
Asymptomatic syphilis	4	6.5	2	3.3	1	1.6	25	40.9	29	47.5	61
Total	61	8.1	170	22.7	56	7.5	337	44.9	125	16.7	749

TABLE 2.—*Incidence of Pupillary Abnormalities in 208 Cases of Dementia Paralytica*

	Cases	Per Cent
True Argyll Robertson pupils	18	8.7
Partial Argyll Robertson pupils	35	16.9
Unproved Argyll Robertson pupils	51	24.6
Total incidence of Argyll Robertson pupils	104	50.2
Other pupillary abnormalities	93	44.4
Normal pupils	11	5.4
Total	208	100.0

Tabes Dorsalis.—In 186 cases of tabes dorsalis the findings were as given in table 3.

The same statement as to the influence of the stage of the disease on the incidence of pupillary abnormalities is as applicable here as it was in the cases of dementia paralytica. Dillmann [19] found in advanced cases of tabes dorsalis that the reaction to light was absent in 76 per cent, whereas in early cases it was absent in only 23.7 per cent. The

18. Bowman, K. M., and Raymond, A. F.: Physical Findings in Schizophrenia, Am. J. Psychiat. **8**:901, 1929.

19. Dillmann, cited by Bumke,[15] pp. 196 and 320.

observations of Leimbach [20] were exactly similar, except that he found the reaction to light absent in 47 per cent of the early cases. The question as to whether the Argyll Robertson pupil is more characteristic of dementia paralytica or of tabes dorsalis seems to us of little significance, as it is present in a large percentage of the cases of each disease. Our cases would seem to indicate that the Argyll Robertson phenomenon is slightly more frequent and absolutely normal pupillary reactions more rare in tabes dorsalis. This is in accord with the literature, particularly with the observations of Faure and Desvaulx,[21] who found normal pupils in only 3.5 per cent of 200 cases of tabes.

TABLE 3.—*Incidence of Pupillary Abnormalities in 186 Cases of Tabes Dorsalis*

	Cases	Per Cent
True Argyll Robertson pupils	31	16.7
Partial Argyll Robertson pupils	9	4.9
Unproved Argyll Robertson pupils	73	39.2
Total incidence of Argyll Robertson pupils	113	60.8
Other pupillary abnormalities	68	36.6
Normal pupils	5	2.6
Total	186	100.0

TABLE 4.—*Incidence of Pupillary Abnormalities in 32 Cases of the Tabetic Form of Dementia Paralytica*

	Cases	Per Cent
True Argyll Robertson pupils	4	12.5
Partial Argyll Robertson pupils	3	9.3
Unproved Argyll Robertson pupils	16	50.0
Total incidence of Argyll Robertson pupils	23	71.8
Other pupillary abnormalities	8	25.1
Normal pupils	1	3.1
Total	32	100.0

The Tabetic Form of Dementia Paralytica.—In this group of 32 cases, in which there was a mixture of the clinical syndromes of tabes and dementia paralytica, the findings were as given in table 4.

Cerebral Vascular Syphilis.—In this group of 66 cases the chief manifestation of the disease was the sudden onset of a focal symptom, such as hemiplegia, hemianopia, aphasia and the like. In these cases the diagnosis was cerebral thrombosis on the basis of a syphilitic infection. Cases of dementia paralytica with focal signs are not included. As a rule, in these cases only slight, if any, evidence of syphilitic meningitis was shown. The findings in this group are shown in table 5.

20. Leimbach, cited by Bumke,[15] pp. 197 and 320.

21. Faure, M., and Desvaulx, cited by Bumke,[15] pp. 199 and 321.

We have not found in the literature any record of series of cases similar to these, and from our observations we conclude that the Argyll Robertson pupil is not common in cases in which cerebral vascular lesions are the only manifestation of a syphilitic infection of the nervous system. This is in accord with the pathologic observations in this type of case, in which it is often impossible to distinguish the changes in the blood vessels from those due to arteriosclerosis. In view of its probable significance in regard to the location and the pathologic features of the lesion causing the Argyll Robertson pupillary reaction, it is interesting to note that in the two cases with the complete Argyll Robertson pupil, there was a lymphocytosis in the spinal fluid of 66 and 23 cells, respectively. This would indicate a certain degree of meningeal and possibly also of parenchymatous involvement.

Syphilis of the Spinal Cord (Exclusive of Tabes Dorsalis).—This group consists of 17 cases in which clinical examination localized the

TABLE 5.—*Incidence of Pupillary Abnormalities in 66 Cases of Cerebral Vascular Syphilis*

	Cases	Per Cent
True Argyll Robertson pupils	2	3.0
Partial Argyll Robertson pupils	1	1.5
Unproved Argyll Robertson pupils	1	1.5
Total incidence of Argyll Robertson pupils	4	6.0
Other pupillary abnormalities	35	53.2
Normal pupils	27	40.8
Total	66	100.0

pathologic process to the spinal cord, and identified it as chiefly in the nature of a thrombosis of a blood vessel of the spinal cord. In this group of cases are included several of early neurosyphilis in the form of neurorecurrence. In none of the 17 cases was the Argyll Robertson pupil found. Minor abnormalities of the pupil were found in 13 cases (76.4 per cent), in 2 of which (12 per cent) the pupillary abnormality was due to previous iritis. That none of the patients showed the Argyll Robertson pupil is of significance in view of Robertson's original idea that disease of the spinal cord is probably the cause of miosis.

Meningovascular Neurosyphilis.—This group is composed of 179 cases of syphilis of the central nervous system in which the clinical signs and laboratory findings were interpreted as indicative of a diffuse meningeal or vascular involvement, and in which a diagnosis of other types of neurosyphilis (dementia paralytica, tabes, etc.) was not tenable. The findings are shown in table 6.

A review of the literature shows that every possible pupillary change can be found in these cases, but the true Argyll Robertson pupil is

relatively rare, and its presence would suggest the possibility of a tabetic or dementia paralytica type of involvement. This does not mean, however, that the Argyll Robertson pupil is pathognomonic of tabes or dementia paralytica, as it has been found in other types of neurosyphilis by Weiler [16] and others. Our records also indicate that the Argyll Robertson pupil can occasionally be found in cases presenting a syndrome that is not clinically consistent with the diagnosis of tabes or dementia paralytica. Since we have no pathologic examination in these cases, and since many of the cases have not been followed long enough,

TABLE 6.—*Incidence of Pupillary Abnormalities in 179 Cases of Meningovascular Neurosyphilis*

	Cases	Per Cent
True Argyll Robertson pupils	2	1.1
Partial Argyll Robertson pupils	7	4.0
Unproved Argyll Robertson pupils	27	15.1
Total incidence of Argyll Robertson pupils	36	20.2
Other pupillary abnormalities	95	53.1
Normal pupils	48	26.7
Total	179	100.0

TABLE 7.—*Incidence of Pupillary Abnormalities in 61 Cases of Symptomatic Neurosyphilis*

	Cases	Per Cent
True Argyll Robertson pupils	4	6.5
Partial Argyll Robertson pupils	1	1.6
Unproved Argyll Robertson pupils	2	3.3
Total incidence of Argyll Robertson pupils	7	11.4
Other pupillary abnormalities	25	40.9
Normal pupils	29	47.7
Total	61	100.0

we cannot exclude the possibility that tabes or dementia paralytica will develop in them. We should agree, however, that the Argyll Robertson pupil is uncommon in uncomplicated syphilitic meningitis or syphilitic involvement of the cerebral vessels, and its presence is probably indicative of parenchymatous involvement. The latter is to be expected on the basis of the anatomic location of the lesion causing the phenomena of the Argyll Robertson pupil.

Asymptomatic Neurosyphilis.—In this group are 61 cases. The patients entered the hospital for various complaints, giving no clinical evidence of neurosyphilis. The diagnosis of syphilis was made by the positive results of an examination of the spinal fluid. The findings in these cases are given in table 7.

In 3 of the 4 cases in which true Argyll Robertson pupils were shown the patients entered the hospital dying, and death was due to syphilitic heart disease. No autopsy was performed in these 3 cases, and since the clinical observation was inadequate, the possibility that they would have shown definite evidence of parenchymatous involvement of the nervous system cannot be excluded. The other case has not been followed long enough to determine whether clinical signs and symptoms of neurosyphilis will develop.

Congenital Syphilis.—Congenital syphilis of the central nervous system in this analysis has not been grouped separately, as the clinical and pathologic findings are the same as in acquired neurosyphilis.

COMMENT ON THE CASES PRESENTED

From a consideration of the 749 cases here analyzed, it is seen (table 8) that the Argyll Robertson pupil was probably present in

TABLE 8.—*Incidence of Pupillary Abnormalities in 749 Cases of Neurosyphilis*

	Argyll Robertson Pupils		Other Abnormalities		Normal Pupils		
	Number	Per Cent	Number	Per Cent	Number	Per Cent	Total
Tabetic and dementia paralytica types	240	56.2	169	39.6	17	4.2	426
Other types	47	14.5	168	52.0	108	33.5	323
Total	287	38.3	337	44.9	125	16.8	749

56.2 per cent of 426 cases of dementia paralytica and tabes dorsalis, and in only 14.5 per cent of 323 cases of other types of neurosyphilis. Our statistics indicate that this type of pupillary reaction is not found at all in syphilis involving the cord (in the form of syphilitic myelitis or thrombosis), and that it is rare in syphilitic cerebral thrombosis and asymptomatic neurosyphilis. Normal pupils were found in only 16.8 per cent of the 749 cases studied. Although Argyll Robertson pupils can occur in every form of neurosyphilis, its presence is indicative of parenchymatous involvement and is of great importance in regard to diagnosis and prognosis.

SUMMARY

1. The Argyll Robertson pupil is defined as one that has the following characteristics: (1) an absence of the reaction to light, (2) miosis, (3) imperfect dilatation in response to instillations of atropine and to painful stimuli, (4) an absence of reaction to vestibular stimulation and (5) an active reaction on accommodation for near objects.

2. These characteristics are due to a destruction of the pupillary light reflex fibers and the sympathetic fibers.

3. The present state of knowledge of these pathways is given, and it is shown that the pupillary light reflex fibers and the sympathetic fibers run together for a short distance in the anterior end of the brain stem just ventrally to the posterior commissure. A destructive lesion at this point would explain all of the phenomena of the Argyll Robertson pupil.

4. Any lesion at this point would produce an Argyll Robertson pupil, but the only incontrovertible cases heretofore reported were associated with syphilis of the nervous system, except a few cases associated with a gliomatous invasion in this region. Therefore, for all practical purposes the Argyll Robertson pupil is pathognomonic of syphilis of the central nervous system.

5. The frequent occurrence of this phenomenon in neurosyphilis is probably due to a localized involvement of the light reflex and sympathetic fibers in the tegmentum by the syphilitic infection.

6. In an analysis of the records in 749 cases of neurosyphilis it was found that the Argyll Robertson pupil had been observed in 287 cases, or 38.3 per cent. The 287 cases were subdivided as follows: the tabetic and dementia paralytica forms of neurosyphilis, 240 cases, or 83 per cent; other forms of neurosyphilis, 47 cases, or 17 per cent.

7. The occurrence of an Argyll Robertson pupil is indicative of parenchymatous involvement, and although it is not absolutely diagnostic of any one form of neurosyphilis, it suggests an involvement of the tabetic or dementia paralytica type.

EPILEPSY

XIII. AURA IN EPILEPSY; A STATISTICAL REVIEW OF 1,359 CASES

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There are many inaccuracies in present knowledge concerning persons who are subject to epileptic seizures. This is due in large part to the absence of an adequate body of statistical data. There has, indeed, been a multitude of reports covering single cases or small groups of patients, but the facts detailed, though valuable in themselves, give no clue as to the relative frequency of the observed phenomena. Information which has been compiled concerning large groups has been drawn almost entirely from institutional patients. In the United States these compose only about 7 per cent of persons subject to epilepsy and are not, therefore, representative. In the nature of the case, institutions house especially those who are physically or mentally incompetent.

Reynolds,[1] in 1861, drew attention to the lack of dependable and proportioned knowledge concerning epilepsy in the following words: "The trustworthy beliefs of the day are compounded with the false notions of tradition as well as with the hasty generalizations of the individual. Now for the purpose of correcting these, or of showing their true value, when they have any, no method is more convincing than the numerical." He published a book containing minutely tabulated data concerning persons with epilepsy, but, unfortunately, had only 81 case reports with which to work.

We believed that data which would most nearly represent a cross-section of epileptic phenomena could best be secured from physicians who see epileptic patients in their offices or in the clinics of general hospitals. Letters were written to the members of the American Neurological Association and of the Association for Research in Nervous and Mental Diseases, asking if they would be willing to make complete reports on patients with epilepsy whom they might see during the following twelve months. They were also asked to recommend other

From the Neurological Unit, Boston City Hospital, and the Department of Neuropathology, Harvard Medical School.

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1. Reynolds, J. R.: Epilepsy, Its Symptoms and Treatment and Relation to Other Chronic Convulsive Diseases, London, J. & A. Churchill, 1861.

physicians who did careful work on patients with epilepsy. Questionnaires were printed and mailed to those who offered to cooperate in this study. Six thousand questionnaires were requested and sent out. Of those returned, 1,069 were usable in our study.[2] These were supplied by 78 different men or clinics. In addition, 458 blanks were filled out for our own private patients and those who came to the clinic of Boston City Hospital. It should be emphasized that this and subsequent communications on the subject are the work of the many physicians who have collaborated in the study. This is the first of a series of papers based on analysis of the records.

The percentage of these extramural patients who had been at some time in an institution corresponds roughly with the percentage of the total number of patients with epilepsy in the country who are at present in institutions. In our group of patients there was, however, some selection owing to the selection of the collaborators. These are neurologists whose clientele consists mostly of adults. Pediatricians and surgeons did not have a full representation.

The study of the aura [3] is in itself interesting and is of potential value in localization. It may indicate the portion of the brain or the neurologic pathways concerned in the seizures. According to Gowers,[4] the term "aura" was first used by Pelops, the master of Galen, who suggested that the sensation of a cold vapor which had been described

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2. The physicians who collaborated and the number of usable blanks returned by each were: the Mayo Clinic (L. H. Ziegler, H. W. Woltman and associates), 239; W. A. Smith, 80; C. D. Camp, 57; A. P. Solomon, 57; Temple Fay, 43; T. J. Heldt, 43; G. H. Hyslop, 38; Andrew C. Gillis, 34; Walter F. Schaller, 34; R. R. Grinker, 29; D. M. Olkon, 26; Max H. Weinberg, 25; Alfred Gordon, 20; J. L. Eckel, 15; Mabel D. Ordway, 13; W. H. Riley, 13, and W. J. Adie, 12. Harold R. Merwarth, J. Notkin, Joshua Rosett and F. G. Ebaugh and associates sent in 11 each.

Those sending from 6 to 10 usable blanks were: E. M. Bridge, E. B. Block, C. A. Patten, Nicholas Gotten, David Nathan, Laurence Selling, H. C. Naffziger, Beverly R. Tucker, W. W. Young, Peter Maybarduk, Ralph Hamill, Marcus Neustaedter and H. A. Riley.

Those sending 5 blanks or fewer were: James B. Ayer, G. B. Hassin, William Hughes, A. G. Kimberley, Forrest N. Anderson, Morris Grossman, I. Leon Meyers, George A. Moleen, D. C. Wilson, C. S. Bluemel, L. H. Cornwall, M. L. Gerstle, Ransom A. Greene, J. C. Michael, D. J. McCarthy, Charles Potts, R. B. Smalley, Lauren H. Smith, Frank T. Stevens, George A. Blakeslee, I. B. Diamond, James H. Huddleson, George P. Sprague, E. W. Taylor, William B. Terhune, John C. White, Lawson Wilkins, G. L. Adamson, Albert M. Barrett, Peter Bassoe, G. L. Collins, J. L. Garvey, R. Finley Gayle, R. L. Glass, G. O. Grain, G. W. Henry, S. R. Leahy, J. E. Moran, Charles A. McKendree, C. A. Noble, Jr., Andrew Rosenberger, M. E. Silverman and Walter Timme.

3. We shall follow usage in employing only the singular form of the word aura.

4. Gowers, W. R.: Epilepsy and Other Chronic Convulsive Diseases. Their Causes, Symptoms and Treatment, London, J. & A. Churchill, 1901.

to him by patients might be wind passing up the veins. The point which we made in the beginning, that a bizarre and unusual occurrence which has been described is often accepted as a usual event, is illustrated by this word. "Aura" has come to represent all premonitory symptoms, but among the 327 different sensations described in this study, not one represents an aura in the original meaning of the word.

It is natural that among physicians of ancient times aura involving the extracranial regions of the body should be looked on as the cause of the subsequent attack. The laity today commonly believe that when there is an epigastric aura "something wrong with the stomach" is to be blamed for the seizure. Three hundred years ago Erastus pointed out that this was not so, that the aura is a part of the seizure.

The tabulation of aura is complicated because many patients describe more than one form; the aura may vary either in its presence or in its character from time to time, and some patients are too young or too much deteriorated mentally to describe their sensations. Also, by a startled look, or a movement toward safety, a patient may indicate that he has a warning of the impending seizure but may be unable afterward to recollect it. It is more accurate to speak of the recollection of aura rather than of their presence. There may be amnesia regarding events which ocurred several minutes before the seizure began. If consciousness is lost late, the aura, instead of being premonitory, will be the commencement of the motor phase of the convulsion. In this study all the aura mentioned have been tabulated, including the few which were evidently prodromal symptoms (present for hours or days) or were objective rather than subjective.

INCIDENCE OF AURA

A warning of the impending seizure has been reported by various authors as being present in from one quarter to one half of their patients. The only series of cases adequate in number is that of Gowers, who found that of 2,013 patients in whom the point was noted (approximately two thirds of the total number studied by him), 57 per cent had an aura.

Among the 1,527 cases reported here, the presence or absence of aura was noted in 1,359, or 89 per cent. An aura was present in 764, or 56.2 per cent, practically the same percentage as Gowers'.

INFLUENCE OF SEX

An aura was reported in 54.8 per cent of 763 men and in 58 per cent of 596 women. The immediate inference is that women are more careful witnesses in the matter of their symptoms. The difference, however, is not great.

INFLUENCE OF ETIOLOGY

The patients were divided into two groups: those with a history of mental abnormality or injury of the brain antedating the onset of the seizures and those without such a history. Fifty-six per cent of the second group (patients with so-called essential epilepsy) had aura, as compared with 54.4 per cent of the smaller group (those with so-called symptomatic epilepsy). This difference is not significant. It is not related to the distribution of incidence according to sex, for the group with essential epilepsy contained a slightly larger proportion of men. It can be accounted for by the fact that those with symptomatic epilepsy showed more mental deterioration.

INFLUENCE OF TYPE OF SEIZURE

Convulsions of the grand mal type presumably involve a larger portion of the brain than those of the petit mal type, and the seizure is much more striking. One might expect that aura would be more frequently noted in grand mal than in other types of seizures. The questionnaires did not provide for complete differentiation on this point. It was possible, however, to ascertain the incidence of aura in patients having only petit mall or equivalents and in those having grand mal (with or without petit mal).

Patients with aura formed 42 per cent of those who had only petit mal or equivalents and 51 per cent of those who had had grand mal. In this tabulation the records which did not state whether or not aura were present were included. They were relatively more numerous in one (the petit mal) group. If these records are left out of account, the incidence for the group having only petit mal is 50 per cent, and for others, 57 per cent.

INFLUENCE OF MENTAL STATE

Gowers suggested that aura are more frequent among intelligent patients than among those who are mentally deteriorated. It was likewise our general impression that the patients with grave mental deterioration who are seen in institutions have aura relatively infrequently. At first glance our tabulation did not seem to bear this out.

Table 1 shows the percentage of patients who had aura.

As will be shown in the following section, the increase in the incidence of aura in patients who were mentally deteriorated is due to the fact that these patients had had seizures for a longer time. The unusual degree of difference in the figures depending on whether or not the blanks in which the incidence of aura was not mentioned are included is due to the fact that the greater the mental defect of the patients the more often was the information concerning aura missing. Apparently

some of those who were markedly defective mentally were children or patients who could not tell whether or not they had had a warning.

The computations in table 1 are inexact because in the case of mentally deteriorated patients the aura noted may have been present in the past (when the patients were normal). Also, the higher incidence of seizures in the mentally abnormal may be due to the longer duration of their symptoms, with the greater possibility of their having experienced aura at some time. To test this point tabulation was made with respect to the number of years during which the seizures had been present.

TABLE 1.—*Incidence of Aura with Reference to Influence of the Mental State*

Mental Condition of Group	Percentage of Group Who Had Aura if the Number of Those for Whom Information as to Incidence of Aura Was Not Given Is	
	Excluded from the Computation	Included in the Computation
Normal	55	53
Slightly deteriorated	57	51
Definitely deteriorated	63	53
Not stated	58	45

TABLE 2.—*Incidence of Aura with Reference to the Duration of the History of Epilepsy*

Duration of History of Epilepsy	Percentage of Patients Who Had Aura if the Number of Those for Whom Information as to Incidence of Aura Was Not Given Is	
	Excluded from the Computation	Included in the Computation
Less than 1 year	49	40
1 to 4 years	48	43
5 to 9 years	62	56
10 years or more	62	56

INFLUENCE OF DURATION OF HISTORY

Table 2 shows the percentage of patients who had aura. It is apparent that there is a distinct difference in the incidence of aura between patients who had seizures for less than five years and those who had them for a longer period. In the latter group the increase over the former group is 27.4 per cent (first column). An undue proportion of the records for those who had seizures for less than a year were incomplete, perhaps because more of these patients were children. The increase in the incidence of aura with the passage of time is presumably due to the greater total number of seizures and the greater likelihood of experiencing aura. The increase in the incidence of aura with the passage of time is more pronounced in the "essential" than in the "symptomatic" group.

With this tabulation in mind, one can conclude that mental deterioration in itself is associated with a decreased frequency, or at least a decreased recollection, of aura.

INDIVIDUAL AURA

In the main, aura were described in the patients' own words. Consequently, a great variety of expressions, 327 in all, were used. Obviously in many instances different descriptions were employed for the same sensation. The more intelligent of the patients gave more accurate, though perhaps not more picturesque, accounts.

In table 3 these descriptions are assembled under general headings. Separation of the patients into the so-called "essential" and "symptomatic" groups is made. In the case of each patient synonyms were eliminated, but each aura that was different was listed. There are 1,059 aura and 750 patients represented in the table. One hundred and sixty-seven patients named two or more symptoms which were not synonyms as their aura.

VARIOUS GROUPS OF AURA

The aura which are listed in table 3 were assembled into groups, and duplications to the number of 43, which occurred within a group, were eliminated. For example, if a patient mentioned both nausea and abdominal pain, he was credited with one aura of the digestive tract. If he mentioned nausea and dizziness an aura was counted under both "digestive" and "dizzy." The percentage distribution of the 1,016 aura was calculated on the basis both of all aura and of all patients with aura (table 4).

Six and seven-tenths per cent of the patients described subjective states which were either pleasant (one-fifth of the cases) or unpleasant (four-fifths of the cases). This, of course, does not include the large number of patients whose premonitory sensations were accompanied or followed by an unpleasant feeling of fear or dread because of the seizure which they knew was impending. The Russian writer, Dostoievsky, described his own aura as a sensation of supreme pleasure: "You healthy young people can't imagine the bliss which we epileptics experience in the moment which precedes the attacks." Hardly one in a hundred of the patients in this study had a sense of pleasure associated with the aura, a fact which speaks against the numerical importance of seizures or their occurrence as a means of escape from a psychologic situation.

Two and two-tenths percent of the patients described disturbances of consciousness. In many instances the sensation was perhaps that of losing consciousness and therefore was the beginning of the seizures

TABLE 3.—*Distribution of Aura Described by 750 Patients, with Reference to Presence or Absence of Evidence of Injury to the Brain Antedating the Onset of Seizures*

Aura	Incidence		
	"Essential" Group	Group with Injury to Brain	Total
Subjective States, Unpleasant			
Fear, apprehension, premonition	20	3	23
Depression	5	..	5
Sensation of death	4	..	4
Startled feeling	2	..	2
Fear of seizure	2	..	2
Fear of falling	1	..	1
Uneasy	1	..	1
Unpleasant	1	..	1
Horror	1	..	1
People approaching from behind	1	..	1
Subjective States, Pleasant			
Happy, exaltation, euphoria	6	..	6
Pleasant	1	..	1
Feels like laughing	1	..	1
Pleasant dreams	1	..	1
Consciousness and Orientation			
Semiconscious, fading, receding	8	..	8
Repeating former experiences	3	..	3
Blankness, lost feeling	2	..	2
Feeling of falling	1	..	1
Sense of having seen	1	..	1
Objects lose relativity	1	..	1
Mental States			
Mind adrift, confused, dazed, dull	27	2	29
Fixed idea	1	..	1
Peculiar mental state	1	..	1
Deviation of thoughts	1	..	1
Feeling of thinking	1	..	1
Dreamy state	1	..	1
Forgetfulness	1	..	1
Amnesia	..	1	1
Repeats words	..	1	1
Makes misstatements	..	1	1
Mood			
Irritable	6	1	7
Excited, talkative, restless	6	1	7
Biting	1	..	1
Talks obscenely	1	..	1
Weeping	..	1	1
Ill-Defined Sensations and States			
Indescribable, peculiar or "funny" feeling	37	6	43
Weakness	26	4	30
Faintness, goneness, sinking, "groggy"	15	4	19
Drowsy, tired	16	2	18
Nervous feeling	12	1	13
Call for mother (children)	5	..	5
Petit mal	4	1	5
Dull or heavy feeling	5	..	5
Sick	4	..	4
Peculiar feeling, localized	4	..	4
Desire to lie down, lazy, sluggish	4	..	4
Like taking anesthetic	2	1	3
Feel spell coming	2	1	3
Shaky	2	..	2
Something rushes to head	2	..	2
Badly	1	..	1
Floating on air	1	..	1
Everything stops in brain	1	..	1
A shade coming down	1	..	1
Sensations of Dizziness			
Dizzy	83	15	98
Vertigo, giddy	14	2	16
Light-headed, things going around	11	..	11
Head spins like a top	2	..	2
Sensations Referred to Head			
Headache or pain	20	4	24
Pressure, throbbing, roaring, noise	13	1	14
Head feels queer	9	1	10
Singing, ringing, grinding, buzzing	4	1	5
Hammer-like, pulsating, snap	3	..	3
Head feels big	2	..	2
Loose feeling, spreading out	2	..	2

TABLE 3.—*Distribution of Aura Described by 750 Patients, with Reference to Presence or Absence of Evidence of Injury to the Brain Antedating the Onset of Seizures—Continued*

Aura	Incidence		
	"Essential" Group	Group with Injury to Brain	Total
Gastro-Intestinal Sensations			
Epigastrium or Stomach			
Peculiar sensation	29	2	31
Fulness, pressure, heaviness, sinking	9	1	10
Faintness, weakness, sickening, goneness	9	..	9
Turning, gas, buzzing, fluttering	6	..	6
Pain	4	..	4
Warming feeling	2	..	2
Pulls back, contraction	2	..	2
Something running around, thrill	2	..	2
Abdomen			
Pain	5	1	6
Sensation, peculiar feeling	5	..	5
Distress	3	1	4
Distention, light feeling	3	..	3
Quivering, pricking, cramp	3	..	3
Epigastrium to Head (or Throat)			
Peculiar sensation, rushing	7	1	8
Gas, bubbles	5	..	5
Wave, lump, choking	4	..	4
Nausea	2	..	2
Intestines			
Gas	1	..	1
Cramps	1	..	1
Other Sensations and Symptoms			
Nausea	19	6	25
Choking	6	4	10
Vomiting	2	1	3
Hunger	2	..	2
Swallowing	2	..	2
Diarrhea, desire to move bowels	2	..	2
Constipation	1	..	1
Passing gas	1	..	1
Sensation in liver	1	..	1
Sensations Relating to Temperature and Circulation			
Chilly, shivering	13	..	13
Palpitation	10	..	10
Heat, flushing of face	1	2	9
Blood rushes to head	8	..	8
Cold (in specified area)	6	..	6
Pallor	3	..	3
Sweating	2	1	3
Hot or cold	2	..	2
Congestion in head or eyes	2	..	2
Cyanosis (hands or lips)	2	..	2
Heat of spine	..	1	1
Wind on back	1	..	1
Sweating in hands	1	..	1
Blood leaves head	1	..	1
Neck feels tight	1	..	1
Sensations Relating to Breathing			
Dyspnea	8	..	8
Air shut off, smothering	4	2	6
Air feels close	..	1	1
Rattling in throat	..	1	1
Salivary Glands			
Asks for water	3	..	3
Dry mouth	3	..	3
Water running from mouth	2	..	2
Special Sensations			
Pain			
Leg or arm	3	1	4
Region of heart	4	..	4
Back or rectum	3	1	4
Face, mouth, tongue	2	2	4
Eyes, eye	2	1	3
Neck	2	..	2
Legs	1	..	1
One-sided	1	..	1
Paresthesia—pins and needles—prickling—tingling			
Arm or hand	6	7	13
Location not named	6	1	7
Face, scalp, head, mouth	3	3	6
Arms or hands	2	2	4
One-sided	1	3	4
Legs	1	..	1
Creepy feeling up spine	1	..	1

TABLE 3.—*Distribution of Aura Described by 750 Patients, with Reference to Presence or Absence of Evidence of Injury to the Brain Antedating the Onset of Seizures—Continued*

Aura	Incidence		
	"Essential" Group	Group with Injury to Brain	Total
Special Sensations—Continued			
Numbness			
Arm, hand, leg	6	11	17
Region not stated	10	1	11
Mouth, lips, tongue, nose, ears	10	1	11
Arms or hands	3	..	3
Head	2	1	3
Extremities	1	1	2
Face	1	..	1
Miscellaneous			
Tongue feels thick or big	4	..	4
Burning in throat or chest	2	..	2
Pressure in eyes	1	..	1
Vision			
Darkness, blindness	13	4	17
Flashes, scotoma	10	2	12
Specks before eyes	11	..	11
Vision blurred	7	..	7
Sees double	4	..	4
Colors	3	..	3
Moving pictures	2	..	2
Sees visions	2	..	2
Sees people	1	1	2
Black and white	1	..	1
Blindness (one eye)	1	..	1
Too many candles at mass	1	..	1
Things appear too near or too far	1	..	1
People move slowly	1	..	1
Something jumping before eye	1	..	1
Light on one side	1	..	1
Black cloud floats to me	1	..	1
Saw second coming of Christ	1	..	1
Staring	..	1	1
White (from left to right)	..	1	1
Hearing			
Ringing, buzzing, strange whistling noises	7	3	10
Auditory disturbance	3	..	3
Sharp sound	1	..	1
Clock ticking	1	..	1
Distressing sound	1	..	1
Music of peculiar rhythm	1	..	1
Music in one ear	1	..	1
Smell			
Disagreeable	6	3	9
Peculiar	2	..	2
Odor of bananas	1	..	1
Camphor	..	1	1
Smell of ironing	..	1	1
Taste			
Bitter	1	..	1
Sour	..	1	1
Abnormal Movements			
Motor Symptoms			
Twitching (portion not stated)	12	1	13
Electric shocks	4	1	5
Twitching or jerking			
Hand, arm or leg	7	5	12
Mouth, face, jaw	8	4	12
Hands, arms or legs	9	..	9
Eyes roll up	3	..	3
Head	3	..	3
Eyelids	1	1	2
Tongue (up and down)	1	..	1
Abdomen	1	..	1
Spasm or stiffening			
Hand, arm or leg	10	11	21
Hands, arms or legs	7	..	7
Pulled to one side—head, eyes	4	3	7
Cry	4	2	6
One-sided face	4	1	5
One-sided	2	1	3
Neck, tongue	2	1	3
Abdominal muscles	1	..	1
Erection of penis	1	..	1
Hiccup	..	1	1
Eyes feel crossed	..	1	1
Paresis			
Unable to talk	3	..	3
Paralysis of legs	1	..	1
Weakness of leg	1	..	1
Weakness of legs	1	..	1
Stumbling, one leg	..	1	1
Total	616	124	1,059

rather than a sensation which preceded it. One might expect more sensations of "things going black" or of "fading away." It is, however, a remarkable fact that persons who breathe nitrogen ordinarily lose consciousness without premonitory symptoms and often do not know after the incident is over that they have been unconscious.

Some of the changes in the mental state described by patients (4.9 per cent of the patients) might be classified as disturbances of consciousness. In the small group in which there were alterations of mood these were presumably in many instances prodromal symptoms noted by the patients' associates. Probably the proportion having

TABLE 4.—*Percentage Distribution of Groups of Aura Among All Aura and All Patients*

Type of Aura	Total Number of Aura	Percentage of All Aura	Percentage of All Patients
Subjective states			
Unpleasant	41	4.1	5.5
Pleasant	9	0.9	1.2
Disturbances of consciousness	18	1.8	2.2
Mental state	37	3.6	4.9
Mood	17	1.7	2.1
Ill-defined sensations	159	15.6	21.5
Dizziness	126	12.5	16.8
Other sensations referred to head	59	5.8	7.9
Digestive tract	140	13.9	18.7
Temperature and circulation	59	5.8	7.9
Respiration	16	1.5	2.1
Salivary glands	8	0.8	1.1
Special sensations			
Pain	21	2.1	2.8
Paresthesias	38	3.7	5.1
Numbness	45	4.4	6.0
Other sensations	7	0.7	0.9
Vision	69	6.8	9.2
Hearing	18	1.7	2.4
Smell	14	1.3	1.8
Taste	2	0.2	0.2
Abnormal movements	106	10.4	14.1
Paresis	7	0.7	0.9
Total	1,016	100.0	135.3

prodromes would have been larger if there had been space on the blanks for the listing of prodromes as distinct from aura.

Approximately one-fifth of the patients had sensations which they were wholly unable to describe, or which they could describe only in general or vague terms. Here, again, some of the sensations, such as "weakness," "faintness," "sinking," may indicate the loss of consciousness or of motor control which marked the beginning of the convulsion. Some intelligent patients who were at a loss for words with which to describe their aura stated that the sensation was distinctly disagreeable or even "horrible."

Naturally enough, the head was the portion of the body most frequently assigned as the site of the aura. The single expression most frequently used was "dizziness." By this term most patients did not mean giddiness or vertigo but had reference to the original

meaning of the word i. e., "stupid" or "confused." A variety of other sensations were referred to the head. Thus one sees that the aura in fully half of the patients concerned the mental state, the general feeling or mood or the head.

The largest group of aura which related to the body was referred to the viscera, especially to the digestive tract (18.7 per cent). The size of this group helps to explain the obstinacy of the belief that a disturbance of the digestive tract is the cause of seizures. The visceral aura arises, as Salmon, Frisch and others have pointed out, in the vegetative centers of the brain—perhaps in the hypothalamus.

So many patients present evidence of disturbance in the functioning of the autonomic nervous system, especially vasomotor changes, and the theory of vascular spasm of cerebral vessels as a cause of certain seizures is so attractive that one scans with interest any case in which the aura includes symptoms of sympathetic or vagal disturbance. Reports have been made of patients whose entire seizure consisted of vasovagal or of sympathetic symptoms. Seven and nine-tenths per cent of the patients studied had aura which signified changes in circulation, in temperature or in activity of the sweat glands. Seventeen patients spoke of the face or head feeling flushed; three, of pallor of the face. These figures will receive further attention when examinations of the patients are detailed in a later paper. Relatively few patients (3.2 per cent) mentioned changes in respiration or in the activity of the salivary glands. To the patients whose symptoms suggest vagal disturbance one should perhaps add many of those listed in the gastro-intestinal group.

Only 2.8 per cent of the patients mentioned pain, although if the 34 patients with pain in the head or abdomen were added the proportion would be approximately 7 per cent. Four patients experienced precordial pain, which suggests that angina pectoris was followed by angina cerebralis.

Certain aura have localizing value to the diagnostician. Knowledge of this subject is limited, because animal experiments cannot be used for the localization of aura and there has been little attempt to correlate aura with cerebral lesions found at autopsy. One scrutinizes with care sensations involving the face or extremities. Eleven and one-tenth per cent of the patients spoke of paresthesias and numbness. In two fifths of these the symptoms affected one side. Disturbances of the special senses are important because they probably denote disturbance of the main receiving stations in the cerebral cortex. The greatest number of patients describing such disturbances mentioned aura involving vision. (9.2 per cent). However, terms such as "darkness," "blindness" and "blurred vision" may be only the means of expressing loss of consciousness. Two and four-tenths per cent of the patients had

abnormal sensations of hearing. Hallucinations of smell were found in 1.8 per cent. Only two patients spoke of a premonitory sensation of taste. Even in these one might question whether a sour or bitter taste might not be due to the eructation of gastric contents rather than to a disturbance of the nerves.

Abnormal muscular movements are of most value in the localization of lesions in the brain causing focal or jacksonian seizures. In such cases the patients are aware of the movements because of the late loss of consciousness. Fourteen per cent of the patients had a warning because of muscular movements. In many instances, however, the movements consisted of dispersed myoclonic jerks, or general spasm, without localizing value. Less than 1 per cent of the patients had premonitory weakness or paralysis. In the three instances in which patients were unable to talk, the difficulty may have been aphasia rather than paralysis of the muscles involved in speech.

TABLE 5.—*Percentage of Various Types of Aura with Reference to the Mental State* *

Type of Aura	Mental Status				Total Number of Cases
	Normal	Slightly Deteriorated	Deteriorated	Not Stated	
Changes in mood or mental state, or ill-defined states	25	30	23	23	26
Dizziness and sensations referred to head	20	16	23	23	20
Gastro-intestinal or abdominal sensations	16	17	21	14	16
Temperature, circulation, respiration	8	11	9	11	9
Pain, paresthesias, numbness	10	8	7	12	10
Vision, hearing, taste, smell	12	8	13	5	10
Abnormal movements or paralysis	9	10	4	12	9

* Only patients without history of injury to the brain antedating seizures and those 10 years of age or older are included. Five hundred and eighty-three patients and 822 aura are represented.

TYPE OF AURA AND ETIOLOGY

If a summary is made of the types of aura encountered in the "essential" and "symptomatic" groups, one finds that aura of probable cortical localizing value (pain, numbness, paresthesia, the special senses of taste, smell, hearing and vision, or motor disturbances) were present in 41 per cent of the patients without a history of injury of the brain antedating the seizures, and in 70 per cent of the patients with such a history.

TYPES OF AURA AND MENTAL STATUS

Data from the blanks were tabulated in order to ascertain whether the types of aura bore any relationship to the patients' present mental status. This computation is somewhat inexact because aura do not necessarily refer to the present time whereas the mental status does. Only patients 10 years of age or older who had no history of injury of the brain were included. The groups of aura were more condensed than in table 4. The results are listed in table 5.

It was found that the differences between the mentally deteriorated patients and the mentally normal patients were not great. The mentally deteriorated patients had a disproportionate number of aura consisting of dizziness and other sensations referred to the head, or visceral sensations and disturbances of the special senses. The patients who were mentally normal had an unusual proportion of sensations referred to the extremities: paresthesia, numbness and abnormalities of movement.

One might ask if the visceral aura so prominent in the mentally deteriorated are due to the more marked pathologic condition of the subcortical areas, and whether the tactile sensations so prominent in the mentally normal are to be explained by more acute observation or by a loss of consciousness which occurs unusually late. Certain aura can be allocated to specific motor or sensory areas of the brain. Others,

TABLE 6.—*Percentage Distribution of Groups of Aura in Cases Reported by Gowers and Those Reported in This Series*

Type of Aura	Gowers, Percentage of Patients	This Series, Percentage of Aura
Unilateral	17.7	8.6
Bilateral, general	4.6	38.2
Visceral	17.7	14.6
Vertigo, etc.	18.7	12.0
Cephalic	8.2	5.6
Psychic	8.3	11.2
Visual	15.9	6.7
Auditory	6.2	1.7
Olfactory	1.1	1.3
Gustatory	1.6	0.1

including the sensation of losing consciousness, can be explained only as an involvement of diffuse areas concerned with association.

COMPARISON WITH GOWERS' CASES

Gowers used a somewhat different grouping of the aura in his cases. Furthermore, he listed only one aura for each patient, presumably the sensation which was first mentioned. There is further difficulty in comparing his figures with ours in that his figures as given do not exactly agree. We arranged the 1,016 aura listed in table 4 to conform with the grouping used by Gowers for his 1,102 patients. A comparison on this basis is not exact for the reasons mentioned. There are, however, wide discrepancies in some of the items (table 6). Gowers' group shows relatively large proportions of patients with aura which are unilateral and which involve the special senses—vision, hearing and taste. These high proportions are balanced in our group by the much greater proportion (38.2 per cent, as compared with Gowers' 4.6 per cent) of patients with bilateral or general symptoms. This difference is perhaps due to the fact that since Gowers listed only one aura for each patient, he

tended to omit those which were least specific. It is possible, of course, that, in spite of the great number of automobile accidents at the present time, traumatic epilepsy was more frequent in Gowers' day than in ours.

CONCLUSIONS

Tabulation was made of the aura in 1,359 noninstitutional patients having seizures as a presenting symptom. Aura were present in 764, or 56.2 per cent.

The incidence of aura was slightly greater in women than in men. There was little difference in the incidence of aura in the "essential" or the "symptomatic" groups.

Aura were less frequent in patients having only petit mal or its equivalents than in others.

The incidence of a history of aura was not less in the mentally deteriorated, because the mentally deteriorated patients had had seizures for a longer period. In those who had had attacks for five years or longer the incidence of a history of aura was 27 per cent greater than in those who had had less than five years of seizures. The mentally deteriorated more frequently had visceral aura, and the mentally normal, aura of pain, paresthesia or numbness.

Descriptions of aura as given by 750 patients are tabulated. Aura of potential localizing value (disturbances of peripheral sensation, paresthesia, numbness, disturbances in taste, vision, hearing or smell, or abnormal muscular movements) occurred in 70 per cent of the patients with a history of injury of the brain antedating the seizures and in 41 per cent of those without such a history.

Gowers' series differed from ours in that a larger proportion of his patients had aura which were unilateral or which involved the special senses.

A COOPERATIVE RESEARCH IN SCHIZOPHRENIA

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Despite the fact that schizophrenia has been known under one or another name for centuries, the nature of the disorder even at the descriptive level remains in no small measure yet to be determined. It is true that there is a voluminous literature describing the multifarious deviations from psychologic normality and no small number of publications dealing with organic variations. But the question is still open as to which of the deviations are essentially characteristic of the psychosis. In the picture as presented by the patient we have to deal with pathologic phenomena of several different categories. The abnormalities may comprise the effects of bad hygiene, the effects of habituation, the effects arising primarily out of the fundamental cause or causes of the disorder and the defects arising from secondary operations of the primary causal factors. The need remains for an adequate definition of the psychosis. This definition should, of course, delimit it from all other disorders; it should be broad enough to include all of the subvarieties; it should include none but characteristic attributes.

The literature on schizophrenia as now available, while profuse in qualitative material, is defective in its content of accurate quantitative information. But an even more significant defect is the lack of homogeneity of the data as they exist.

These data have been obtained by many investigators using different technics on a variety of patients living under dissimilar environmental conditions. They are of correspondingly limited utility for the purpose even of formulating a technically satisfactory definition. In large part they are entirely unusable for the type of studies of correlation that

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is needed for the analysis of the problem as a problem. There is need, then, for the collection of an extensive body of descriptive data on a fairly large number of patients studied under uniform conditions. This topic has been discussed at greater length in another communication.[1]

While it is true, as has been emphasized elsewhere, that the mere enumeration of traits is a research method of strictly limited utility, it is likewise true that attempts to carry out research on a problem that has not been accurately set are likely to result in numerous ineptitudes.

Even at the descriptive level, certain substantial accomplishments can be hoped for. At this level can be studied the question as to whether schizophrenia represents a disease entity or whether, like fever or headache, it represents a variety of essentially different disorders. The detection of disease entities has not infrequently been accomplished at this level. For example, myxedema was so discovered. In principle, this sort of discovery amounts to the detection of consistent syndromes.

Schizophrenia may well represent a variety of syndromes. Given a sufficient body of homogeneous data, such syndromes should be detectable by the use of systematic statistical methods. The detection of a syndrome, however, does not in itself afford any evidence of causality. To continue the previous illustration, the mere fact that deficiency of the thyroid gland was discovered as a characteristic concomitant of myxedema served merely to delimit the problem of causality. The causal relationship of the defect of the thyroid gland to the disorder had to be proved experimentally. This was done in two ways. Surgical removal of this gland for goiter was shown to be followed by myxedema, and the symptoms of myxedema were shown to be ameliorated by the administration of thyroid substance.

In the case of schizophrenia the approach to the problem of causation will presumably necessitate three steps. The accumulated data must be tested, as indicated, for the existence of characteristic syndromes. The items making up the syndromes may stand in casual, consequential or concomitant relationships to each other. The next step is to test empirically for consistency of concomitance. It must be determined which features of the syndrome vary *pari passu* with changes in the clinical condition of the patient. In principle, this method serves to differentiate the essential from the fortuitous elements in the picture. Finally, among those features that vary in step with the clinical condition, causation may be sought.

For the detection of causation we have in principle two empiric methods of procedure. One is to make frequent cross-sectional determinations of the physiologic and psychologic characteristics of the patient,

1. Hoskins, R. G.: An Analysis of the Schizophrenia Problem from the Standpoint of the Investigator, J. A. M. A. **97**:682 (Sept. 5) 1931.

ideally before and during the onset of the psychosis, or, less satisfactorily, during an attack and through a remission of the psychosis. For this aspect of the research, the most valuable type of patient is one who passes rather promptly through episodic acute attacks, with approximate normality between exacerbations. Thus the events might be placed in temporal sequence. Those invariably occurring early may be expected to include the causal factors of those arising later. The second method is the experimental. This amounts in effect to the arbitrary augmentation or reduction of any suspected factor and noting the consequences as mirrored in the clinical state of the patient. Commonly, therapeutic studies, when based on rational "leads," have a certain value in the determination of causation. Without further elaboration of the philosophic aspects of the problem, suffice it to state that these are the two general methods by which etiology is commonly determined. This paper embodies the description of an experimental project that has been evolved in the light of the foregoing considerations.

In the earlier years, the studies consisted of a series of tests of the vital functions, psychologic tests and rather conventional psychiatric studies.[1] As the work progressed, more and more difficulty was experienced in interpreting the data because of a rather high degree of variability among the test findings of the individual patients. In 1931, it was determined to concentrate attention for the time being on variability as such. A so-called seven months' plan was formulated, and the work of the entire staff for a year was devoted to carrying it out. The plan has been briefly described elsewhere.[2]

The purpose of the plan was to collect observations on a considerable number of physiologic, psychologic and psychiatric variables for about seventy-two schizophrenic patients. This required four weeks of testing for each patient; each was then given two months' rest during which, however, studies of the action of certain pharmacodynamics and of the spinal fluid were made. The tests were then repeated and another period of rest interposed. Finally, in the seventh month the tests were made again. Thus each patient was studied rather elaborately three times without the complication of treatment other than unavoidable situational influences.

The entire environmental setting was kept as constant as possible, though absolute constancy is impossible in a hospital for mental illness. For example, every significant change in the clinical state of any patient constitutes a change in the environment of the others.

The schedule of studies is indicated in the accompanying table. An attempt was made to conduct the study under conditions that would

2. Hoskins, R. G.: The Worcester State Hospital Research Project in Schizophrenia, J. Nerv. & Ment. Dis. **75**:663 (June) 1932; Arch. Neurol. & Psychiat. **28**:454 (Aug.) 1932.

First Week

Monday:	9:00 a.m. Psychometrics	1:00 p.m. Physical and psychiatric examinations
Tuesday:	9:00 a.m. Psychometrics	1:00 p.m. Physical and psychiatric examinations
Wednesday:	9:00 a.m. Psychometrics if necessary	1:00 p.m. Physical and psychiatric examinations
Thursday:	7:30 a.m. "Basal metabolism," including rectal temperature, pulse rate, blood pressure, weight and height; sample of blood collected for quantitative analysis and phytotoxic test 9:30 a.m. Breakfast	1:00 p.m. Psychiatric examination
Friday:	7:30 a.m. "Basal metabolism" 9:30 a.m. Breakfast	1:00 p.m. Diagnosis by admitting staff; psychiatrist's note
Saturday:	7:30 a.m. "Basal metabolism"	p.m. Mental note

Second Week

Sunday:	a.m. Rest	p.m. Rest
Monday:	9:00 a.m. Experimental psychology	1:00 p.m. Psychiatric observations in ward 3:00 p.m. Photography—two nude poses
Tuesday:	9:00 a.m. Experimental psychology	1:00 p.m. Psychiatric observations in ward
Wednesday:	9:00 a.m. Experimental psychology if not previously completed	1:00 p.m. Psychiatric observations in ward
Thursday:	8:00 a.m. Oculocardiac test 9:30 a.m. Breakfast	1:00 p.m. Psychiatric observations in ward 1:30 p.m. Dental examination and roentgen studies of skull, chest and gastro-intestinal tract
Friday:	a.m. Blood volume, plasma volume, hemoglobin, blood gases, blood p_H, blood morphology	3:00 p.m. Schneider test of vasomotor efficiency; psychiatric observations in ward
Saturday:	8:00 a.m. Period of rest; recheck of Friday's observations if necessary	p.m. Rest

Third Week

Sunday:	7:00 a.m. Start 24 hour collection of urine	p.m. Collection of urine
Monday:	7:00 a.m. Finish collection of urine 8:30 a.m. Inject phenolsulphonphthalein; collect specimens	2:30 p.m. Blood pressure
Tuesday:	7:00 a.m. Start collection of 24 hour urine 7:30 a.m. "Basal metabolism," lung volume 9:30 a.m. Breakfast	1:00 p.m. Psychiatrist's note
Wednesday:	7:00 a.m. Complete 24 hour collection of urine 8:30 a.m. Inject phenolsulphonphthalein, collect specimens	
Thursday:	5:00 a.m. Galactose tolerance control sample 6:30 a.m. Collect samples for chemical analysis of blood and blood counts 7:00 a.m. Galactose tolerance test	1:00 p.m. Psychiatrist's note
Friday:	5:00 a.m. Repeat galactose tolerance test	1:00 p.m. Psychiatrist's note
Saturday:	5.00 a.m. Repeat galactose tolerance test	1:00 p.m. Psychiatrist's note

Fourth Week

Sunday:	a.m. Rest	p.m. Rest
Monday:	8:00 a.m. Fluoroscopic gastro-intestinal studies begun; psychiatrist's note; internist's note on physical status	p.m. Gastro-intestinal series
Tuesday:	a.m. Gastro-intestinal studies continued; psychiatrist's note	
Wednesday:	Gastro-intestinal studies continued 8:00 a.m. Blood sedimentation test; bromsulphthalein; test for hepatic function; psychiatrist's note	
Thursday:	Gastro-intestinal studies continued	p.m. Psychiatrist's note

secure the maximum utility of the data for purposes of correlation. Especially was it desired to secure observations of the psychiatric status coincident with the application of physiologic tests that might be modified by the emotional condition of the patient. In this we were not always successful owing to the limitation of personnel, but in general the psychiatric and other observations were so timed that major defects in the data were avoided.

METHODS

The various items are mostly self-explanatory. Standard methods were used throughout. A history of the case was obtained, as discussed in a later section. A thorough physical examination of each patient was made, partly by way of eliciting data bearing on constitutional diagnosis, but especially for the purpose of detecting organic disease that would serve as a fortuitous complication of the study. Any patient showing evidence of disease beyond such passing afflictions as colds or minor ailments was excluded from the series. In short, our aim was to confine the study to subjects free from significant organic disorders.

Patients were included for study only when the research psychiatric staff was able to classify the cases as schizophrenic beyond a reasonable doubt. Patients were excluded on the following grounds: (1) over 50 years of age (chiefly because of the liability to organic changes frequently occurring with advancing age); (2) coexisting disease discoverable by the usual diagnostic methods; (3) adequate histories unobtainable; (4) marked language difficulties; (5) inclusion of feeblemindedness with the schizophrenic picture; (6) symptoms predominantly those of psychoses other than schizophrenia; (7) recent chronic alcoholic or other intoxication.

For purposes of further description, the activities of the various cooperating departments will be dealt with separately.

ORGANIC STUDIES

The determinations of the basal metabolic rates were made on Thursday, Friday and Saturday of the first week and on Tuesday of the fourth week. The patients were taken to the laboratory without breakfast and were put to bed for thirty minutes before being tested. After the period of rest the oxygen consumption was measured for two six minute intervals by the Benedict-Roth method. The results were calculated according to the Harris-Benedict and the Aub-Dubois standards, and the mean of the two values was taken as the basal rate. If superficially "satisfactory" conditions of basality were not secured, the tests were recorded but marked with an asterisk and not subsequently used for statistical purposes. Extreme care was taken to exclude technical error. The machines were tested daily for leakage and for failure to absorb carbon dioxide due to spent soda lime. Tests were recorded as "satisfactory" only when the patient remained quiet during the period of rest and showed no overt evidence of tension during the tests. If, as was usually the case, two consecutive tests differed significantly in the rate of oxygen consumption, the lower was regarded as more likely representative and was used for subsequent studies of correlation. The details of the study (on a large group) and a critique of results have been published elsewhere.[3] After the rate

3. Hoskins, R. G., and Walsh, Anna: Oxygen Consumption ("Basal Metabolic Rate") in Schizophrenia: II. Distributions in Two Hundred and Fourteen Cases, Arch. Neurol. & Psychiat. **28**:1346 (Dec.) 1932.

of oxygen consumption was determined, the patient's weight, standing height, sitting height, rectal temperature and blood pressure (systolic and diastolic) were determined. The results of the last mentioned study have also been reported.[4]

Front and lateral full length photographs were made of each patient in the nude at a standard distance of 6 feet (1.8 meter) and before a coordinate screen. A standard mercury vapor lamp served for illumination.

Oculocardiac Test.—The degree of vagus irritability was determined by a quantitative oculocardiac test. The pulse rate was recorded before and during the application of varying grades of pressure to each and to both eyes through an oculocompressor, following the Roubinovitch method.[5] The pressures used were 125, 150 and 175 mm. of mercury.

As a supplement to the physical examination a thorough dental examination was made. If indications appeared, x-ray plates were made of the teeth.

Roentgenograms.—X-ray plates were made as a routine measure of the skull—lateral and frontal exposures—the chest and the abdomen and pelvis after a barium sulphate meal. A consultant roentgenologic specialist interpreted each plate as to the condition of the sella turcica, sinus, lungs, stomach and intestines.

Weight.—This was obtained with the patient unclothed. By comparison with the Metropolitan Life Insurance Company height-weight-age tables, corrections having been made for shoes and clothing, a nutritional index was calculated.

Blood.—The volume was determined by the congo red method.[6] In brief, a known volume of 1.5 per cent solution of congo red equal in milliliters to one fourth of the patient's weight in kilograms was injected intravenously, after 10 milliliters of blood had been removed for the preparation of the standard. After mild exercise for three minutes and before the end of six minutes a second 10 milliliters of blood was taken. Five milliliters of the blood was added to each of two graduated centrifuge tubes containing 1 milliliter of 1.6 per cent oxalate solution. The control sample of blood was treated in the same way. All four tubes were capped with heavy finger cots and centrifugated for thirty minutes. At the end of this time the total volume and the corpuscle volume were read for each tube. The concentration of the dye in the serum was determined by reading in a colorimeter against a 1:200 dilution of the dye containing the same amount of serum taken from the control tubes. The volume of the plasma was then calculated, and by dividing this value by the percentage of plasma in whole blood the volume of the whole blood was determined.

The hemoglobin was determined in grams per hundred milliliters by means of the Haden-Hausser hemoglobinometer. The value of 15.6 Gm. per hundred milliliters was taken as normal.

Samples of blood were taken from the radial artery and the median basilic vein for determination of the gases and the p_H. The samples were taken directly in glass syringes to which a finely powdered sodium oxalate and sodium fluoride mixture was added, and then the syringes were capped. All venous samples were taken at least thirty seconds after removal of the tourniquet to allow for reestablish-

4. Freeman, H.: The Effect of "Habituation" on Blood Pressure in Schizophrenia, Arch. Neurol. & Psychiat. **29**:139 (Jan.) 1933.

5. Roubinovitch, J.: Presentation of a Manometric Oculocompressor, J. Nerv. & Ment. Dis. **52**:385 (Nov.) 1920.

6. Rowntree, L. G., and Brown, G. E.: The Volume of the Blood and Plasma in Health and Disease, Philadelphia, W. B. Saunders Company, 1929, p. 202.

ment of the normal circulation and to prevent stasis. Exposure to air was completely prevented by handling the blood in the syringes in which the samples were taken. Evidence that this technic was satisfactory will be reported in detail in a subsequent paper.

The arterial and venous oxygen and carbon dioxide were determined in a Van Slyke manometric apparatus. The p_H of the arterial and the venous whole blood was determined by the glass electrode and the electron tube potentiometer of Stadie.[7] All measurements were for whole blood at 38 C.

The morphologic study of the blood included a red cell and a leukocyte count, and a differential count on 200 cells. Both the conventional and the Schilling criteria were used.

The vasomotor efficiency was studied by the method of Schneider.[8]

Renal Function.—The rate of excretion of phenolsulphonphthalein was determined for two of the three test periods, but was then discarded as not significant for patients on whom extensive analyses of the blood and urinalyses were being made.

The urine was collected for two twenty-four hour periods in each of the three test months. At least two and in some cases all of the specimens were obtained by catheterization at the beginning and the end of the collection, with voluntary voidings in between. The patients were under surveillance throughout the entire time of the collection. The volume and specific gravity were measured, and from these values the total solids were calculated. Each specimen was centrifugated and the sediment subjected to microscopic examination. Qualitative tests were made for indican, albumin, sugar, bile, acetone and urobilinogen. An aliquot portion of each sample was analyzed for total nitrogen, urea, uric acid, creatinine and ammonium nitrogen. The residual nitrogen was determined by subtracting the sum of all the determined bodies from the total nitrogen.

Volume of the Lungs.—This was determined by the use of a spirometer in which was measured the total forced expiration after a forced inspiration. In psychotic patients this datum is significant when the volume is equal to, or above, prediction. Subnormal values are obviously of dubious significance because of the dependence of the datum on the cooperation of the subject.

All samples of blood for chemical analysis were taken in the morning before breakfast. The nitrogen of the urea, amino-acid, uric acid, creatinine and creatine fractions was determined, and this was subtracted from the nonprotein nitrogen to give the residual nitrogen. Except for the urea nitrogen, which was obtained by the method of Looney,[9] and the uric acid, which was at first determined by the method of Benedict and Franke, all determinations were made by the methods of Folin. Cholesterol was determined in the whole blood by the method of Myers and Wardell and blood sugar by the method of Folin.

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7. Stadie, W. C.; O'Brien, Helen, and Laug, E. P.: Determination of the p_H of Serum at 38° C. With the Glass Electrode and an Improved Electron Tube Potentiometer, J. Biol. Chem. **91**:243, 1931.

8. Schneider, E. C.: Further Observations on a Cardiovascular Physical Fitness Test, Mil. Surgeon **52**:18 (Jan.) 1923.

9. Looney, J. M.: The Determination of Blood Urea Nitrogen by Direct Nesslerization, J. Biol. Chem. **88**:189, 1930.

10. Looney, J. M., and Macht, D. I.: The Relation Between the Undetermined Nitrogen of the Blood and Its Toxicity to Lupinus Albus Seedlings, J. Biol. Chem. **63**:60, 1925. Herz, E., and Weichbrodt, R.: Die Toxizität des Serums und ihre Deutung, Deutsche med. Wchnschr. **1**:1210, 1924.

Phytotoxic Index.—As previous studies [10] had demonstrated that the serum of certain psychotic patients was toxic to the growth of seedlings of Lupinus albus, the "phytotoxic index" was included in the study. This index is obtained by dividing the average elongation of the root of the seedling of Lupinus albus in a solution of mixed salts [11] to which 1 per cent of blood serum has been added by the elongation obtained in the same concentration of the same solution alone. From 10 to 25 seedlings were used for each test. The results of this study will be published in detail later.

Sedimentation of the Blood.—The rate was determined by the method of Rourke and Ernstene.[12] This, with the Schilling index, was relied on to supplement the ward observations of the nurses and internists in the detection of intercurrent infections. As a further precaution, morning and evening rectal temperatures and resting pulse rates were recorded as a routine measure each day and the weight once a week.

In the routine studies, standard methods were used throughout. These will be discussed more fully in subsequent reports on the different phases of the research. Suffice it here to state that the more difficult procedures were carried out by thoroughly trained chemists and physicians and the other procedures by well trained technicians under constant supervision. The work was checked at frequent intervals by carrying out the technical procedures in duplicate so that the analytic error in all determinations was kept to the minimum for the specific test.

Pharmacodynamic Tests.—In addition to the routine studies, each patient was subjected to special pharmacodynamic tests during the interval between routine test periods to determine the functional condition of the autonomic nervous system. The details of these tests will be reported separately.

Galactose Tolerance.—The test of Rowe [13] was used as an index of carbohydrate metabolism.

Gastro-Intestinal Function.—Roentgenographic studies included pictures of the abdomen or abdomen and pelvis immediately and at six and twenty-four hour intervals after the ingestion of 180 Gm. of barium sulphate. Plates were made with the patients in the supine position. Fluoroscopic examinations were made with the patients in the erect position. The final progress of the barium through the tract was determined by fluoroscopic examinations repeated daily until evacuation was completed. In connection with the gastro-intestinal studies, each patient was reexamined by a physician to exclude intercurrent physical disorders that might render the findings noncharacteristic. Likewise, each received a special examination by the psychiatrist each morning of the tests as a control on the emotional factor.

Hemato-Encephalic Barrier.—This was tested for permeability by Hauptmann's modification of Walters' bromide method, as used by Malamud and co-workers.[14]

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11. The mixed salts include: 10.4 cc. of a 0.5 molar solution of calcium nitrate, 30 cc. of a 0.5 molar solution of magnesium sulphate and 36 cc. of a molar monopotassium acid phosphate mixed with enough distilled water to make 1 liter.

12. Rourke, M. D., and Ernstene, A. C.: Method for Correcting Erythrocyte Sedimentation Rate for Variations in Cell Volume Percentage of Blood, J. Clin. Investigation **8**:545 (June) 1930.

13. Sleeper, F. H., and Hoskins, R. G.: Galactose Tolerance in Dementia Praecox, Arch. Neurol. & Psychiat. **24**:550 (Sept.) 1930.

14. Malamud, W.; Fuchs, D. M., and Malamud, N.: Barrier Between the Blood and the Cerebrospinal Fluid: I. Changes in Permeability in Mental Diseases, Arch. Neurol. & Psychiat. **20**:780 (Oct.) 1928.

The test consists in feeding the patient bromide for several days and then estimating the ratio between the bromide in the serum and that in the cerebrospinal fluid from the color produced by gold chloride.

Hepatic Function.—Tests were made by the methods of McClure for the determination of cholesterol, bile pigments and bile acids in the bile obtained from duodenal drainage after stimulation with oleic acid. Bromsulphalein determinations of hepatic function, the van den Bergh reaction, the icteric index and Graham tests were also made.

Forty-eight patients were studied before and after the feeding of tyrosine for changes in the phenol content of the blood and urine in relation to the uric acid of the blood. These studies were highly technical, and further discussion of them will be deferred until the data are systematically reported.

Control studies on various features were made on a group of physicians, medical students and laboratory workers.

PSYCHOLOGIC CONTRIBUTION TO THE STUDY

For its part of the study, the psychologic department used a battery of tests and experimental procedures aimed at obtaining samples of the psychologic and psychophysiologic characteristics of the patients. These ranged from reflex responses through various motor and intellectual functions to complicated personality traits and attitudes. The nature of these can be seen from the list of items to be given. The attempt was made to touch on a fairly comprehensive group of functions in order to enable us to study the intercorrelations of these and their connection with the physiologic and other characteristics studied during the same period. It was also desired to study the variability in these functions over the period.

In this paper we shall not discuss the possible effects of previous examinations, the order of items in the psychologic schedule and the effect of other, nonpsychologic studies made in the same period. The consideration of such problems must be left for future papers, as must also the discussion of the question of controls, the attitude of patients and examiners and other problems.

It might be stated, however, that the general program was so arranged as to have the psychologic part of the study carried out at times when there was the least likelihood of the results being affected by other procedures, especially those often unpleasant to the patient, such as blood tests. The psychometric examinations came at the very beginning of the month of study, in the mornings and afternoons of the first two days of the first week. If the examination was for some reason or other not completed during the first two days, the third day of the week was used for this purpose. During the second week the major part of the experimental psychologic procedures were gone through on the corresponding days of the week. After the first period of study, additional experimental procedures were introduced, but because of the fulness of the schedule it was necessary to place these in what was called the "zero" week. This was the week preceding the first week of repeated study, i. e., the last part of the last week of the two month period of rest.

The schedules used are herewith presented. They were modified in only a few cases. The figure in parenthesis preceding the item refers to the number of times it was, as a general rule, given during the seven month period.[15] After each

15. A number of items were for one reason or another not given the requisite three times, usually because the nature of the test or experiment precluded it.

item is given the reference to the sources for the technics so far as it is possible to give them.[16]

Psychometrics

Week I: First Day

(3) K-S clinical formboards [17]

(3) Stanford-Binet test [18]

(3) Otis S. A. intermediate test (alternate forms) [19]

(3) S-K symbol-digit test [17c]

(3) Writing test [17c]

(3) Drawing test [20]

(1) Freyd occupational interests [21]

Second Day

(3) Worcester 2C formboard [22]

(3) Army Alpha (alternate forms) [23]

(3) G. E. pinboard [24]

(3) Kent-Rosanoff association [25]

(1, 2) Thurstone [26] or Bernreuter [27] personality inventories

(1) Heilbronner test [28]

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16. A number of the technics are original and will in time, it is hoped, reach publication. These are indicated. A number of modifications of existing technics have also been made. They will be reported in the papers on the specific subjects.

17. (*a*) Shakow, D., and Kent, G. H.: The Worcester Formboard Series, Pedagogical Sem. **32**:599, 1925. (*b*) Kent, G. H., and Shakow, D.: A Graded Series of Formboards, Personnel J. **7**:115, 1928; (*c*) Unpublished material.

18. Terman, L. M.: The Measurement of Intelligence, Boston, Houghton Mifflin Company, 1916, p. 362.

19. Otis, A. S.: Otis Self-Administering Tests of Mental Ability: Intermediate, Forms A-D, Yonkers on Hudson, N. Y., World Book Company, 1928. Footnote 17*c*.

20. Goodenough, F. L.: Measurement of Intelligence by Drawings, Yonkers on Hudson, N. Y., World Book Company, 1926, p. 177.

21. Freyd, M.: The Measurement of Interests in Vocational Selection, J. Person. Research **1**:319, 1922-1923.

22. Bronner, A. F.; Healy, W.; Lowe, G. M., and Shimberg, M. E.: A Manual of Individual Mental Tests and Testing, Boston, Little, Brown & Company, 1927, p. 174. Footnote 17*c*.

23. Yoakum, C. S., and Yerkes, R. M.: Army Mental Tests, New York, Henry Holt & Company, 1920, p. 303. Wells, F. L.: Army Alpha—Revised, Personnel J. **10**:411, 1932.

24. (*a*) O'Connor, J.: Born That Way, Baltimore, Williams & Wilkins Company, 1928, (*a*) p. 213; (*b*) ibid., p. 217. (*c*) Footnote 17*c*.

25. Kent, G. H., and Rosanoff, A. J.: Association in Insanity, Am. J. Insanity **67**:37 and 317, 1910. O'Connor.[24b]

26. Thurstone, L. L.: A Neurotic Inventory, J. Soc. Psychol. **1**:3, 1930.

27. Bernreuter, R. G.: The Personality Inventory, Stanford University, Calif., Stanford University Press, 1931.

28. Heilbronner, K.: Zur klinischpsychologischen Untersuchungstechnik, Monatschr. f. Psychiat. u. Neurol. **18**:115, 1905. Footnote 17*c*.

(1) Memory test [29]

Experimental Schedule

Week II: First Day

(3) Steadiness [30]

(3) Prod [31]

(2) Knee jerk [32a,c]

Second Day

(3) Pursuit [33]

(3) Reaction time: simple visual, choice visual, simple auditory [17c]

(3) Tapping [17c]

(2) Galvanic threshold [34]

Third Day

(1) Luria "Motorik" [35]

"Zero" Week

(1, 2) Observation [36]

(1) Interruption [37]

(1) Rorschach [38]

On a few occasions it was necessary to change the regular psychometric schedule owing to foreign language difficulty or to lack of cooperation. Under these circumstances tests such as the following were substituted: Army Beta, Witmer cylinder, Witmer formboard, ship, Kent-Kohs color cubes, Woodworth-Wells substitution, Porteus mazes and Healy picture completion.

PROGRAM OF PSYCHIATRIC STUDY

The psychiatric study of patients on the research service was made in accordance with an established schedule. Admission to the service followed a diagnosis of schizophrenia by the ward physicians and a recommendation as to suitability. On admission the patient was assigned to one of the four staff psychiatrists for

29. Wells, F. L.: Mental Tests in Clinical Practice, Yonkers on Hudson, N. Y., World Book Company, 1927, p. 170. Footnote 17*c*.

30. Dunlap, K.: Improved Forms of Steadiness Tester and Tapping Plate, J. Exper. Psychol. **4**:430, 1921. Footnote 17*c*.

31. Miles, W. R.: Personal communication to the authors. Footnote 17*c*.

32. (*a*) Benedict, F. G.; Miles, W. R.; Roth, P., and Smith, H. M.: Human Vitality and Efficiency Under Prolonged Restricted Diet, Washington, D. C., Carnegie Institution, 1919, p. 155; (*b*) ibid., p. 176. (*c*) Footnote 17*c*.

33. Koerth, W.: A Pursuit Apparatus: Eye-Hand Coordination, Psychol. Monog. **31**:288, 1922. Footnote 17*c*.

34. Footnote 17*c*. Benedict, Miles, Roth and Smith.[32b]

35. Lebedinsky, M. S., and Luria, A. R.: Die Methode der abbildenden Motorik in der Untersuchung der Nervenkranken, Arch. f. Psychiat. **87**:471, 1929. Footnote 17*c*.

36. Lewin, K.: Kilmaufnahmen über Trieb und Affektäusserungen psychopathischer Kinder (verglichen mit Normalen und Schwachsinnigen), Ztschr. f. Kinderforsch. **32**:414, 1926. Footnote 17*c*.

37. Ovsiankina, M.: Die Wiederaufnahme unterbrochener Handlungen, Psychol. Forsch. **11**:302, 1928. Footnote 17*c*.

38. Rorschach, H.: Psychodiagnostik, Bern, Ernst Bircher, 1921, p. 174.

thorough study to determine the reaction type. An extensive mental examination, consisting of an elaboration of the Kirby schema, was made over a period of four days. On the fifth day the psychiatric findings, together with social service history and the results of the pertinent psychologic examinations, were presented to the diagnostic staff, consisting of four psychiatrists, three or more internists and two psychologists; both of the latter groups were well versed in practical psychiatry. After an adequate discussion of the findings and an interview with the patient, classification was made as to diagnosis, subtype and suitability for further study as determined by the criteria cited earlier in this article. In the classification of the cases of the patients into various subtypes of schizophrenia, the kraepelinian system was used for clearcut types; otherwise the cases were designated as "mixed," "unclassified" or "late indeterminate."[39] The terms were regarded as merely roughly descriptive, not as connoting nosologic differentiation. Unanimity of opinion as to subclassification was not considered essential. When once accepted as suitable, throughout the rest of the period of study the patient was interviewed daily by the psychiatrist, and careful daily notation was made of his mental state and behavior, especially in relation to the procedures of study. In the second and third periods of study, precisely the same order of psychiatric routine was followed. This included even the diagnostic procedure, in order to insure a reasonable validity of diagnosis and to correct any errors made. During the periods of rest no intensive psychiatric study was conducted, but the patient was seen daily and often interviewed by the psychiatrist. The information was recorded in the form of weekly notes. In order to supplement the material obtained directly by the psychiatrists, a daily conference between the nurses and the psychiatrists was held at which an account was given of each patient's activities. The nurses and attendants also recorded independently the usual ward notes in accordance with a standard established by the psychiatrists. Further, under the direction of the chief psychiatrist, two ward observers took notes on selected aspects of the patient's behavior.

STATISTICAL ASPECTS OF THE STUDY

During the progress of the study about five hundred thousand quantitative observations were made. Means had to be adopted to insure the recording and organization of the material. The achievement of four principal aims was undertaken: (1) assuring from the outset of the project the statistical validity of the data; (2) safeguarding the completeness of the data; (3) consolidating the records emanating from various departments; (4) analyzing and presenting the results.

It was believed that a statistical department should not defer functioning until after the figures had been produced, but should take an active part in devising the experiments and the record forms, thus making it more probable that an ultimate statistical analysis would really lead to a clarification of the problems in question. Consequently the statistician and his staff were in constant contact with the various research workers, discussing with them such points as numerical requirements, control groups, control of interfering variables and experimental

39. The term "late indeterminate" was used to designate the chronic condition in which differential diagnostic features had disappeared.

and observational errors. Each subproject was "set up" in preliminary outline, considering: (*a*) the objects of investigation, (*b*) the justification of the project, (*c*) the methods to be utilized, (*d*) the records and reports required, (*e*) enumeration of the personnel and its duties, and (*f*) the estimated cost of the undertaking.

Such outlines were submitted by each research worker to the various members of the group, including the statistician, for criticism before starting to work on the problem.

The problem of safeguarding the completeness of the data necessitated a comprehensive checking system to make certain that all reports due from the various workers were received.

The consolidation of the mass of experimental data coming from many sources constituted one of the major practical problems in the statistical set-up. Our purpose was to combine facilitation of statistical analysis with accessibility of the data to the various workers. We wished to present the raw data in a form which permitted quick scanning and which would give a longitudinal and cross-sectional picture of a patient at a glance. Obviously a coding system would not serve this purpose. Besides, we were working with an unusually large number of variables but a relatively small number of patients on whom not single but frequently repeated observations were made at short intervals. Thus the conventional punch-card system was not applicable.

A special system of consolidating and recording was devised. For each of the major branches of the research a separate soft paper card, 10½ by 6 inches (25.4 by 15.24 cm.), was used. The cards had twelve lines, providing space for observations over one year, the number of columns depending on the number of variables involved in the study to which the card referred. One card with twenty-seven headings was devoted to social history; one card with fourteen headings dealt with the chemical composition of the urine. The chemical composition of the blood and the blood counts were recorded on two cards with a total of forty-two headings; one card with ten columns contained the data on basal metabolic tests. Psychometry and phychologic experiments appeared on three cards, with a total of fifty-five columns. Miscellaneous clinical data relating to the temperature, the pulse rate, the weight, the gastro-intestinal studies and the galactose tolerance were given on one card of twenty-six columns. Therapeutic data were recorded on one card under twenty-nine headings. This card included, in addition to results on medication, data on physical activity in terms of hours spent in work and play, the number of visitors received by the patients and the like. Lastly, there was a card on which the positive findings of the physical, dental and roentgen examinations were recorded.

At the bottom of each card a descriptive title summarized the kind of data to which the card was devoted. The cards were placed in overlapping arrangement, showing the bottom label, on a division sheet and bound in a so-called "straight-post visible binder," from which either the whole case or a single card could be taken out at any time with the greatest facility.

The binder had a capacity permitting the inclusion of the history of each of thirty-three patients for twenty-five years and giving space for the records of five

hundred variables. A given month was always kept in the same horizontal line on all cards, so that by placing cards side by side paired values for correlation studies could be easily picked. It should be mentioned that the transferring of the data to these consolidation forms was subjected to a triple check. Simultaneously with this consolidation scheme all observations were coded, and as soon as a period for a given patient was completed, the coded values were scored into frequency tables.

This system made it possible to compute descriptive averages such as arithmetic means and standard deviations at any given phase of the study. Frequency distributions of each variable were prepared according to the following scheme: distributions of all values per period, distributions of individual mean values per period, distribution of individual ranges per period, distribution of individual ranges over all periods and distributions of the differences of individual means by signs between two periods.

Besides this, in instances in which the number of observations warranted, as for example in the case of bodily temperatures and pulse rates, a separate distribution was prepared for each patient. Thus, for these two and a few other variables we obtained measures of variation for individual subjects, and from these individual standard deviations we prepared frequency distributions of this constant.

Approximately six thousand frequency distributions were plotted, and their constants computed. This furnished an ample basis for the determination of variation within and among subjects. Besides this, an analysis of seasonal trends was attempted for each variable.

The correlation of the many factors constituted the greatest part of the labor in the statistical offices. Many gross correlations were computed and the relationships presented in the form of scatter-diagrams.

Contingency tables were frequently compiled as need demanded.

Finally, the statistical material on many of the different aspects has been assembled, and interpretative memoranda have been prepared.

SUMMARY

A cooperative project for the investigation of schizophrenia in its several aspects is described.

The functions of the laboratories devoted to organic and psychologic features of the research are outlined.

The methods by which psychiatric observations were made is discussed.

The recording and analytic methods used in dealing with the data are recounted.

The project has proved to be practicable and productive.

Clinical Notes

A CASE OF HUMAN BRAIN DEFECT: FAILURE OF THE OPERCULA TO COVER THE LEFT INSULA

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REPORT OF CASE

Clinical History.—J. E. R., a white man, aged 50, had a stroke and died on his way to the hospital. Investigation revealed that he had a sister, but she could not be reached. He had been a drifter. The body was examined in the department of anatomy of the University of Arkansas School of Medicine.

Autopsy.—The brain was removed in the routine manner, shortly after completion of the embalming. The appearance of the specimen is shown in the figure. No evidence was found in the skull that there had been an injury in the temporal region. The defect did not resemble a lesion from injury or disease. The surface of the cortex had a normal appearance. By examination of the basal view of the brain in the figure (*A*) the normal right side can be contrasted with the defective left side. The left temporal pole is definitely restricted in its anterior extent, while the rolled edge of the frontal cortex does not extend as far posteriorly as on the right. A gap is left between the frontal and the temporal lobe, and the insula is exposed in the depth of the gap. In the figure, *B*, a view of the left side of the brain, demonstrates the extent of the exposure of the insula. Both *A* and *B* show the depression to be deep, with abrupt walls. The edges are rounded and smooth.

COMMENT

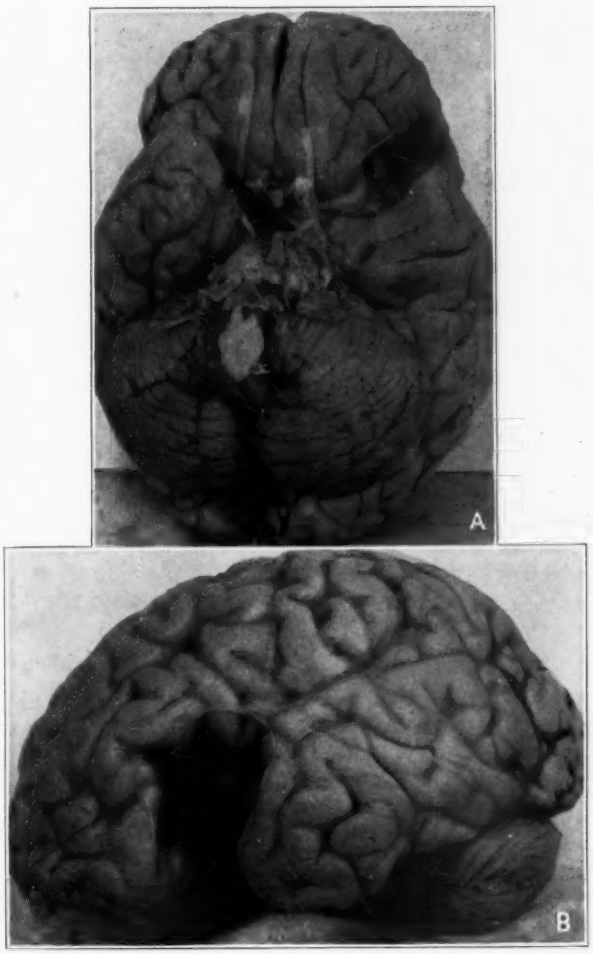
The appearance of the left side of the specimen suggested the condition in a fetus of 7 months, as shown in the Kollmann figure, which is copied in various textbooks on embryology and elsewhere. In the size of the cerebral hemispheres and in the number of sulci the two are very different. But in the failure of the opercula to cover the insula, the specimen represents a retardation of development at approximately that present in a fetus of 7 months.

To make this defect clearer, the story of the development of the cortex in the sylvian region will be told briefly, based essentially on the account given by Bailey and Miller.[1] Early in fetal life, a shallow depression appears on the smooth lateral surfaces of the cerebral hemispheres. This is known as the fossa sylvii. It marks the beginning of the formation of the lateral, or sylvian, fissure. At the bottom of the fossa lies the insula, the part of the cerebral cortex superficial to the corpus striatum. This portion of the cortex lags behind in development, and recedes from the surface as the surrounding cortex develops more rapidly. The boundary walls of the depression thus formed are divided into opercula. In the book of Bailey and Miller,[1] the division is into three opercula: orbital on the

From the Department of Anatomy, University of Arkansas School of Medicine.

1. Bailey, F. R., and Miller, A. M.: Text-Book of Embryology, New York, William Wood & Company, 1929.

anterior side, frontoparietal on the superior side and temporal on the inferior side. Keibel and Mall's [2] description differs from this in that the anterior side is subdivided into frontal and orbital opercula. The temporal operculum extends upward and forward, and the frontoparietal downward. These two begin early and change the direction of the lateral fissure from an oblique to a more horizontal



A, base of the brain; *B*, lateral view of the left cerebral hemisphere.

angle. The orbital operculum covers the anterior part of the insula during the first year of life.

Thus, in terms of embryology, the defect represents an arrested development of the opercula. In the specimen it may be seen clearly that the temporal and

2. Keibel, F., and Mall, F. P.: Manual of Human Embryology, Philadelphia, J. B. Lippincott Company, 1912, vol. 2, p. 102.

orbital opercula are retarded. The retardation of the frontoparietal operculum is less definite. The insula is exposed as in a fetus of 7 months.

It would be of interest to correlate this developmental defect with the clinical findings. As it included the motor speech area, observations on the manner of speech would be of special interest. Unfortunately, such correlation is not possible. Hospital records were consulted, but none for this man were found. Apparently he had not been a patient in any of the hospitals of the city. He was not known to have been examined by a physician while living. A few of his acquaintances were questioned and gave vague answers. His speech was described as "queer," but in just what way they could not make clear; they could understand him. With those at his rooming house he was reserved, being especially noncommunicative concerning his personal affairs.

Apparently this developmental defect is rare. At least, no report of a case in an adult was found in the literature. G. Elliot Smith [3] described an anomaly in this region, but it was in a fetus. In his case the anterior limiting sulcus of the insula was prolonged upward in a manner similar to its homolog in the ape.

D. J. Cunningham [4] described the brain of a chimpanzee in which he demonstrated the exposed insula; he contrasted the condition with the submergence in the normal human brain. Bailey and Miller [1] stated that the change in the angle of the lateral fissure by the growth of the opercula is characteristic of the human brain in distinction from that of the apes. Thus the exposure of the insula in this specimen can be considered as apelike as well as representative of a developmental retardation.

SUMMARY

The case is presented for its interest and for its apparent rarity. Because of the retardation in development of the opercula, the insula was exposed on the left side. This defect is not only developmental but apelike. The significance is not discussed, because it was not possible to obtain a history adequate for possible correlation.

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3. Smith, G. Elliot: Note on an Exceptional Brain Presenting Pithecoid Abnormality of Sylvan Region, J. Anat. & Physiol. **38**:158, 1903.

4. Cunningham, D. J.: The Insular District of the Cerebral Cortex in Man and Man-Like Apes, J. Anat. & Physiol. **32**:11, 1897.

SPECIAL ARTICLE

TRAINING OF THE NEUROLOGIST

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TRANSLATED BY R. KEMEL

It would be impossible to overemphasize the importance of the evolution or rather the transformation of the methods and procedures of neuropathology that has taken place in recent years. Carried away by the movement, one does not always realize the advances and changes in the ways of thinking, understanding and acting that have been introduced in the study of neurology. Therefore, it is an excellent thing that the editors of the ARCHIVES OF NEUROLOGY AND PSYCHIATRY have taken the initiative in what may be called a "collective examination of conscience" in order to determine the best methods of education and instruction to be adopted for those who desire to become qualified and specialized neuropathologists, as well as the training that must be given students of medicine who will become general practitioners.

Although the latter have no special inclination toward the study and treatment of diseases of the nervous system, it is absolutely necessary that their education on this point be perfected in order to enable them to recognize at least the nature of the morbid manifestations and to refer these to their real cause; in other words, to determine whether there is present an organic alteration or a functional disorder of the nervous system. It may be added that today, when the pathology of the vegetative system permeates the whole field of medicine, nobody can be a clinician or a therapeutist if he does not possess thoroughly precise data on sympathicopathology as well as on endocrinology.

In order to formulate a judgment on what should be the modern instruction and education of the neurologist it is wise to glance backward and to consider what neuropathology was fifty years ago. At that time many disorders of the nervous system had already been described, and the gross framework of neurology was established, but one was hemmed in by morphologic and anatomicoclinical types and by methods originated by Duchenne (of Boulogne), Charcot and Vulpian, to mention only French neurologists. To be sure, owing to the disorder that existed in the time of Charcot and Vulpian, it was necessary to create landmarks, syndromes based solely on clinical observations and on the data of pathologic anatomy; Charcot did this successfully.

At the time of the death of Charcot the education of the neurologist was clinical and anatomic, but no more. Neurologists did not study the

fine points of physiology, and neurosurgery, which was just born, did not seem to promise the results obtained today.

It is strange that the physiologic way of thinking of Hughlings Jackson was not then known even to neurologists with the broadest information; von Monakow's ignorance of the ideas of the great English neurologist is typical in this regard.

Today no neurologist thinks only as a morphologist, as in the days of Charcot. A radical change has been brought about under the influence of the physiologist; the mode of thinking is physiologic. Functional alteration has relegated consideration of the lesion to second place. This transformation, which appears revolutionary, necessarily had its counterpart in changing the methods of scholastic instruction and education. But that is not all; if it is well to think physiologically, it is better to think biologically. The scientific horizon expands, and the disease is no more considered only as an individual accident, but as a lesion, the cause of which must be sought in the atavistic past of the patients and the consequences of which must be followed through the descendants. Here appears the future social part of neuropathology.

Medicine is not a contemplative science; its aim is to discover the immediate causes of diseases and to treat their manifestations. Consequently, alongside of the biologic tendency appear today prophylactic and therapeutic tendencies; these must be taken into account in methods of instruction.

WHAT THE NEUROLOGIST MUST KNOW

It can never be sufficiently reiterated that the neurologist must have a thorough knowledge of general medicine and the specialties connected with neuropathology, particularly ophthalmology, otorhinolaryngology, syphilology and, last but not least, psychiatry.

Evidently, the neurologist cannot hope to rival the otologist or the ophthalmologist, but he must be capable of examining a labyrinth, of discovering vestibular reactions, of examining the ocular fundus and of discovering ocular paralysis.

As to mental functions, it seems to me just as imperative for the neurologist to be at the same time a psychiatrist as it is for the psychiatrist to possess a refined knowledge of neurology. It must be admitted that this is not always the case, to the great disadvantage of patients and of the progress of mental medicine. Mental medicine has been retarded in its methods and conceptions because psychiatrists are not well versed in neurology and biology; this is one of the reasons why the data offered by psychiatry so often appear precarious and uncertain. The great psychiatrists were all excellent neurologists and often eminent physicians. Today, more than at any other time, it is important that the neurologist possesses a good psychiatric education.

Consequently, the problem of the education of the neurologist appears complex and difficult, and the difficulty increases if one considers the practical instead of the theoretical side of the question. As I have tried to show, the education and instruction of the neurologist must be based simultaneously on physiology, biology and psychopathology, as well as on clinical observation and pathologic anatomy. On what problems should the future neurologist first train his mind and concentrate his attention?

Anatomy and Physiology.—It is evident that a problem of neurology should not be broached without knowledge of the structure of the nervous system or without exact data on the physiology of the cerebrospinal axis; but is it necessary that the student have a thorough knowledge of anatomy and physiology before studying in the clinic? I do not think so. The training given to him by the usual obligatory lectures in anatomy and physiology certainly allows him to enter on clinical studies, which must remain the fundamental basis of the education of the neurologist.

But here it is necessary to be precise. For too many years the anatomic education of the future neurologist has been too theoretical. The structure of the cerebrospinal axis has been taught by means of schemas which in reality deviated too much from the truth. In this way, when a well instructed neurologist was confronted with concrete realities he found himself completely at a loss. It is not alone necessary to do away with these crude schemas or to limit their number; it is also imperative that the student stand on the solid ground of indisputable reality from the time he takes his first steps in neurology. In other words, he must be made to examine personally macroscopic and microscopic preparations of the brain and the spinal cord, and he must be familiar with the various aspects of sections of the brain in different planes. Thanks to this anatomic training, the neurologist will be enabled to visualize a lesion suggested by the symptoms observed.

Anatomic knowledge not only is necessary for the everyday needs of the clinic but is also indispensable for the understanding of physiologic facts. More so than in any other organ, the physiologic functions of the nervous system are imprinted in its anatomic and histologic structure. Obviously, I do not mean that it is possible to conclude what is the function of any part of the cerebrospinal axis from its structure; on the other hand, it is impossible to understand the mechanism of a nervous function if one does not know the anatomic basis. Morphology and physiology seem to me absolutely inseparable in neurology, and, therefore, the young neurologist must necessarily obtain instruction on the structure and the function of the nervous system simultaneously.

Clinical Neurology.—Medicine is altogether a science of observation. Neurology in particular demands thorough and attentive observation as

well as a rigorous method. It is consequently essential that a student in neurology be given a chance to make observations at the bedside, to describe them and to discuss his conclusions before his teachers. In this way he will learn to see clearly, to introduce order among his observations, to bring to light the principal facts, and, above all, to know how to deduce certain pathologic phenomena and to appreciate their symptomatologic value.

His apprenticeship must be served in a clinic which is well provided with selected patients, such as is offered by a faculty of medicine. The clinical service of the Salpêtrière (Professor Guillain) may be cited as an example of this view. It is vain to try and fix the same period of instruction for everybody. Undoubtedly, one or two years are not enough to make the student a neurologist.

Pathologic Anatomy.—Although pathologic anatomy is not much liked by recent neurologists, research into morphologic alterations of the nervous system remains an excellent training, provided it is well directed. Undoubtedly, pathologic anatomy today cannot be considered as the ultimate aim of an observation; biologic research, which is already being carried on, will dominate the future.

Even if histopathology, which has been called ironically "the science of dead things," is not accepted as an end but as a means, it is obvious that this teaching helps one to avoid many errors and pitfalls by holding the imagination in leash and guiding it back to reality. The patient study of anatomic changes will improve the precision of mind and sense of method, will clarify the connections and the limits of so-called morbid entities and will often be the origin of a fruitful hypothesis relating to the pathogenesis and origin of a syndrome. The examples of dementia paralytica, multiple sclerosis and the epidemic encephalitis of von Economo are sufficiently demonstrative. Anatomicopathologic studies must be made at the same time as the clinical studies. The laboratory alone leads nowhere, and a neurologic clinic deprived of precise anatomic observations is an illusion.

Psychiatry.—Armed with a neurologic, clinical and anatomic technic, the young neurologist must extend his knowledge into psychiatry, and for that purpose should serve for at least six months in a specialized service where he may become familiar with the psychoses, as well as with the psychoneuroses which he will have frequent occasion to treat in practice.

In recent times there has been a tendency, not yet extinct, to consider the neurologist more as a physician for organic nervous diseases than as an expert capable of following and adjusting himself to the deviations of the psychoneuroses and their symptomatology, which are all the more disconcerting because they lack an anatomic basis. This

idea, to my mind, is altogether faulty and dangerous. It is faulty because too many examples show its inaneness; and it is dangerous because, if admitted, it delivers psychoneurotic persons to physicians who lack sufficient neurologic knowledge and are incapable of discovering minimal symptoms indicative of organic changes, or worse still, it delivers them to so-called psychotherapeutists who lack medical training and give full rein to their imagination and even to their instincts.

In what clinical section of the hospital can the neurologist learn the treatment for psychoneuroses and psychopathic conditions, the gravity and social repercussions of which do not justify commitment? First, in the neurologic clinic of which I have spoken; it must contain an outpatient department or a policlinic to which unselected patients are admitted. Among these there will be cases of organic disease and a large number of pure psychoneurotic cases. Here the neurologist can study upheavals caused by slight traumatisms of the peripheral and central nervous systems, and familiarize himself with occupational accidents, in which there are so often psychopathologic and organic elements. In fact, it seems to me evident that medicolegal appraisement should be confided to the neurologist rather than to the surgeon or the psychiatrist.

The student will also find rich material for study in the "open services" of psychiatry, which are accessible to everybody and are consulted by numerous patients. The neurologic clinic of a university should contain a service for treatment of slight mental trouble. All who are familiar with the clinic of the Salpêtrière, in which Charcot entrusted treatment for the psychoneuroses to Prof. Pierre Janet, know how much they learned by attending the small wards in which are hospitalized patients with conditions that now are designated as more or less patent schizoid, cyclothymic and hebephrenic states, and patients with hysteria.

Relations with General Medicine.—From the views expressed, it is evident that the neurologic clinic of a university is a vast organization, comprising a clinical service, an outpatient department, a service for the study and treatment of psychoneuroses, specialized services of neuro-ophthalmology and otoneurology, laboratories for research in pathologic anatomy and biology and a service of neurosurgery.

Before going further, however, one must consider the important question of the relations between neuropathology and general medicine. On a solution of this delicate problem depends in part the activity and the recruiting of the neurologic clinics of the universities and the successful evolution of neuropathology.

Actually, the clinical service of the faculty, as well as the services of hospitals directed by qualified neurologists, possesses a personal attendance of patients. The patients come to the neurologic service on the

advice of the attending physician, in response to an obscure instinct, on the advice given by friends or sometimes simply by chance. Although the clinical service in which the young neurologist will be trained contains many patients with subacute and chronic diseases, many acute diseases, especially those of infectious origin, will not be seen because these diseases are attended to in the services of general medicine or of pediatrics. I have particularly in mind the cases of acute poliomyelitis in children, epidemic cerebrospinal meningitis, diphtheritic polyneuritis, encephalitis due to measles, varicella and vaccination, and some other diseases.

Undoubtedly, patients suffering from these contagious diseases should be isolated. The service of the neurologic clinic does not contain isolation rooms, but why not provide them? Would it not be advisable in the interest of the patients to hospitalize those with the nervous diseases of which I have spoken, to make diagnoses and to treat them in the service of neurology instead of isolating them in a nonspecialized clinic directed by a physician who has no inclination toward neuropathology? To this there is one objection: Is there no risk of encumbering the neurologic clinic with patients in whose cases the diagnosis is uncertain and who in reality are not suffering from diseases of the nervous system? This argument against the thesis I defend is not without value or foundation, but it is easy to guard against the inconveniences offered by a very large attendance. Would it not be possible to give to the professor directing the neurologic clinic the "right to requisition" from the general service and to admit to his service certain interesting or demonstrative cases of acute infantile poliomyelitis, cerebrospinal meningitis, posteruptive encephalitis and nervous complications of diphtheria? This right of requisition is possessed by the professor of psychiatry of the Asile Clinique Ste. Anne in connection with the admission service of Ste. Anne, through which pass all the mental cases of the district of Paris, and it is due to this that the psychiatric clinic of Paris is well provided with patients suffering from acute or chronic disturbances representing all the different types of mental medicine.

Syphilis.—Considering the frequency of nervous complications of infection by the spirochete, it is highly desirable that the neurologic clinic should receive from the service for syphilis a sufficient number of patients to allow the young neurologist to complete his instruction on this capital point in a minimum of time.

THE NEUROSURGEON

The considerable advances made in surgical treatment of disorders of the nervous system, thanks to the persevering efforts, audacity and

expertness of Harvey Cushing, Frazier, Dandy and Adson, have brought on an increasing complexity in the instrumental and operative technic to such a point that today no general surgeon would try without special preparation to discover an intracranial or medullary neoplasm. In this way, the extremely proper conception of the neurosurgeon has become progressively established. What training must the neurosurgeon receive? This problem is serious in its consequences and comes up for discussion everywhere.

First, one point seems to be established: The neurosurgeon must necessarily have a clinical, physiologic and anatomic training in neurology. It is true that the pioneers of neurosurgery, Jaboulay, Fedor Krause and Chipault, were primarily surgeons to whom the neurologist indicated the region where the operation should be done and explained the topography of the lesion; at times it was the neurologist who suggested the *modus operandi* to the surgeon. This method is no longer accepted, and it is essential that the neurosurgeon be just as capable as the qualified neurologist of making a precise diagnosis and establishing the possibilities of surgical intervention; it is on his decision alone that the choice of operation must depend in any given case. It is through his neurologic training that the neurosurgeon will be able to appreciate the indications of postoperative reactions and so to avoid or mitigate them. But a neurologic training demands several years, and under the circumstances it is permissible to ask whether the neurosurgeon must necessarily be a surgeon before becoming a neurologist. Well known examples (O. Foerster, C. Vincent) demonstrate that the neurologist may become a perfect neurosurgeon. Yet one should not make a rule of an exception. Thierry de Martel, who in France was the initiator of the modern technic of neurosurgery and to whom are due new operative methods as well as a continuously perfected instrumentation, comes from the field of general surgery; although he has acquired long since a neurologic training from Babinski, he remains primarily a surgeon. Personally, I am inclined to think that preparation in major surgery is not a disadvantage but a solid asset for the neurosurgeon. Besides, if the neurosurgeon actually limits himself mostly to interventions on the central nervous system and leaves operations on the peripheral nerves and on the sympathetic to less specialized surgeons, it is probable that this will not long remain so. Neurosurgery must extend its domain to the whole peripheral and autonomic nervous system. To my mind it is too easily forgotten that, in order to perform an operation on some part of the vegetative system, there are needed not only the qualities that make a precise and audacious surgeon but also a perfect knowledge of the data of physiology to enable him to appreciate the results of an exeresis or of a nerve section. In my opinion, the neurologist must be a physiologist as well as an anatomist.

THE NEUROBIOLOGIST

As I stated in the beginning of this article, there has been for several years an increasing tendency to impel the neurologist to enlarge his conceptions, to take a broader view than in the past and to consider facts not only from the anatomicophysiologic angle but also from the biologic aspect. Function and structure are no longer examined individually on one plane, but in their connections with the data of the past and the possibilities of the future. Cerebral anatomy loses its rigidity and immutability and becomes in a certain measure alive and pluridimensional, as the psychiatrist would call it; physiology, pushing the study of the function to its utmost limits, is more than ever before intimately related to the physicochemical and mathematical sciences.

Neurology is full of the concepts of colloids, ions, electric charges, electric potentials, mathematical relations and definite functions. One need only recall the ever-increasing importance of the relatively recent concept of chronaxia. All this leads one to think that in the near future the neurologist will need to assimilate mathematical and biologic knowledge not possessed by the neurologist of former times. It may be reiterated, however, that I do not say that the study of this knowledge should ever displace clinical research. In my opinion, clinical research will always remain the basis of the education of the neurologist; yet this does not mean that the neurologist should not try to surpass this point of view and to see things not only as a physician but also as a biologist.

Since the work of Hughlings Jackson, the concepts of level of disintegration of function, escape from control and chronogenous localization (von Monakow) have been introduced in neurology and are now well established. However, is it possible to assimilate and realize the value of these terms if one does not possess a knowledge of the functions attributed to organisms less advanced than that of man, as well as of precise data on the development of nervous functions in the child?

Complete instruction in neurology must consequently include a purely biologic division; this can be attempted fruitfully, however, only when the neurologist possesses all essential knowledge in morphology, in clinical medicine and in physiology.

The biologic point of view is the most advanced point in the education of the neurologist; he will obtain it only after having analyzed patiently the elemental facts and after having understood their real value; then his outlook will extend to the boundaries that limit the conditions of the most humble relations of life and those that comprise the life of the mind.

Obituaries

SIR PERCY SARGENT
1873–1933

In the death of Sir Percy Sargent, Great Britain has lost her most brilliant neurosurgeon of the postwar period. Sir Percy was born in Clifton, England, in 1873, and matriculated at St. John's College, Cambridge, where he decided on a career in medicine. On going to London, he took an examination for a university scholarship at St. Mary's, but he was so certain that he had no chance of winning the award that he immediately took a similar examination at St. Thomas'. To his surprise, he was successful in both and eventually chose the latter hospital.

At about this time, Sir Victor Horsley was doing the first pioneer work in neurosurgery at the National Hospital, Queen Square. In time he found that the volume of the work was too great to be handled by himself and his associate, Sir Charles Ballance. Consequently, in 1906, Sargent, who had already secured a surgical post at St. Thomas', went to Queen Square as assistant to Horsley to begin his work on the surgery of the nervous system. During the next eight years, until the outbreak of the World War, he served as assistant at Queen Square and at the same time developed into a clever operator in general surgery at St. Thomas' Hospital, where he advanced up the surgical ladder by the inexorable process which is apt to make rank and age synonymous in the large London hospitals.

During the war, Sargent's services were notable. He turned his attention exclusively to surgery of the nervous system with brilliant results. His capacity for quick decision and rapid, skilful execution made him the ideal neurosurgeon at the front, where there was often neither time nor facility for slow, meticulous operating and where twenty—not one—operations on the brain might have to be performed in a day. His service in France was distinguished further by fearless criticism of what he deemed to be inefficient methods of handling cases of injury of the nervous system, a criticism which was not without result. He was eventually appointed consulting surgeon, to the British Expeditionary Forces, for diseases of the nervous system. During this time his work with Dr. Gordon Holmes was a fine example of cooperative effort between a brilliant neurologist and a dexterous surgeon.

Since Horsley had died during the war, Sargent returned to Queen Square to share with Mr. Donald Armour the position of senior surgeon. He then resumed the almost impossible double rôle of neurosurgeon and

general surgeon. As a general surgeon, his brilliance was well recognized at St. Thomas' Hospital and throughout London. But his interest and attitude, like his early training, were always those of a practical surgeon rather than a neurologist. He found no time for research in the physiology and anatomy of the nervous system, as his master, Horsley, had done. Could he have renounced his large practice in general surgery and specialized in the field of neurology after the war, he might well have enriched neurosurgery as greatly as Horsley did.

As it was, he continued in the Horsley tradition without much contact with the work of other neurosurgical clinics. He executed best those maneuvers which called for rapid dexterity and which demanded a minimum of painstaking work at closure. Consequently, his field of greatest success was perhaps surgery of the spinal cord, although he did work of importance on the surgery of the brain and pituitary body.

Sargent's attitude toward the scientific problems of neurology was always one of penetrating criticism. This is well shown in his polished address [1] on epilepsy delivered on the occasion of his induction into the presidency of the Section of Neurology in the Royal Society of Medicine. "A Jacksonian fit," he said, "is merely a series of fractional movements devoid of purpose and determined in point of sequence only by the relative anatomical position of the cortical areas by which they are represented." This is a definition which would have met with the approval of his elder colleague, Hughlings Jackson. In introduction to his remarks on the surgical therapy of epilepsy, he said characteristically, "The surgeon who blindly removes a button of bone places himself on the level of the practitioner of the stone age."

His interests were not limited entirely to the fields of neurology and surgery. He took time for the social and friendly side of life. It cannot be said of him, as it can of many men who succeed brilliantly in their profession, that he failed miserably in the art of living. He was an active worker in numerous charities and had a sustained enthusiasm for the work of the Masonic Lodge, in which he achieved an exalted rank.

Sargent's death will be sincerely mourned by neurologists and neurosurgeons the world over. He was always a charming companion and could be, when the occasion demanded, a witty after-dinner speaker. His gay, bantering manner and graceful presence endeared him to those who were fortunate enough to know and to appreciate the warm heart and the distinguished mind of this great English surgeon.

WILDER PENFIELD, M.D.

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1. Sargent, Percy: Brain **44**:312, 1921.

Abstracts from Current Literature

THE HISTOLOGIC AND PARASITOLOGIC ANALOGIES BETWEEN EARLY CONGENITAL SYPHILIS AND METASYPHILIS: A CONTRIBUTION TO THE PATHOGENESIS OF DEMENTIA PARALYTICA. ERNST STRÄUSSLER, Jahrb. f. Psychiat. u. Neurol. **48**:376, 1932.

In dementia paralytica the brain not infrequently shows lesions that are most atypical of this disease. These atypical lesions are remarkable because they are apparently more closely related to the spirochetes; i. e., they are more specifically syphilitic than the typical lesions of dementia paralytica. Another remarkable feature of these atypical changes is their almost exclusive limitation to the dementia paralytica form of acquired syphilis as well as to the lesions observed in the early stages of acquired syphilis. These atypical lesions are: the so-called colloid or hyaline degeneration of the brain, the miliary gumma, the so-called miliary necrosis and the miliary abscesses of the cerebral cortex.

Alzheimer was the first to describe colloid degeneration of the brain; he found it in two cases, one in a patient with dementia paralytica and the other in a young person with cerebral manifestations of unknown etiology. Since then, only thirteen cases with this type of lesion have been recorded in the literature; in twelve of these the brain showed evidences of dementia paralytica, and in one (Kufs' case) the lesions were those of an "old syphilis cerebri." It would thus seem that dementia paralytica, above all, offers the most fertile soil for the development of colloid cerebral degeneration. As to the miliary gummas, it is significant that miliary gummas in the cerebral cortex not related to gummatous involvement of the meninges occur only in dementia paralytica. No such lesions have ever been observed in the early stages of acquired syphilis. According to the author, the miliary necroses of Hauptman, Herschmann, Schob and Jahnel are closely related to massive collections of spirochetes. They have never been observed in lesions of cerebral syphilis which precede metasyphilis. Miliary abscesses were first described by Schob, in 1925, who observed them in the cerebral cortex in addition to miliary necroses in a case of dementia paralytica. The abscesses consisted of circumscribed collections of leukocytic deposits confluent with similar but less sharply outlined collections of leukocytes. They bore no relation to the miliary necroses. Within the abscesses there was found a network of spirochetes in whose meshes numerous collections of leukocytes could be seen. The abscesses appeared to have been produced by a secondary invasion of leukocytes into a mass of spirochetes. It is noteworthy that such lesions have also never been observed in cerebral syphilis.

Sträussler points out that miliary necroses and abscess-like miliary lesions are never observed in acquired syphilis during the stages preceding metasyphilis. They bear a close relationship to accumulations of spirochetes and are analogous to the miliary syphilomas found in other organs, especially in the liver in cases of congenital syphilis. This, however is not the only analogy between congenital syphilis and metasyphilis. There is little difference between the pathologic process of syphilis congenita praecox, as contrasted with syphilis congenita tarda, and that of acquired syphilis. P. Schneider recognizes four forms of syphilis congenita praecox: (1) syphilis of the stillborn, (2) syphilis of the newly born, (3) syphilis of nurslings, and (4) "recidive syphilis." The last form occurs between the second and fourth years of life and gradually merges with syphilis congenita tarda. In the first group the entire organism is affected by an overwhelming invasion and multiplication of spirochetes. Pathologists generally attribute this intense spirochetal invasion to the inability of the young organism to muster the proper defensive mechanism necessary to cope with the parasite in question. It is not until the organism has become mature that the defensive mechanisms are aroused:

this is particularly the case in infants, especially in those that have survived eight days; in them the number of parasites is diminished, and the degree of tissue reaction increased. At first the syphilitic changes in the viscera and bones become unusually marked, and a nonspecific interstitial and productive inflammatory process develops in the parenchymatous organs which is the basis for the later appearing syphilomas. Marked cutaneous and mucous membrane manifestations generally appear only in those infants that have survived. In this connection it must be emphasized that the typical large, nodular gummatous swellings are unusually rare in syphilis congenita praecox.

As far as the spirochetes are concerned, it may be stated that it is only in dementia paralytica that masses of spirochetes are found in the brain that are at all comparable to those found in congenital syphilis. They are almost never found in the brains of patients with cerebral syphilis. In dementia paralytica the unusually abundant spirochetosis is associated with a nonspecific type of inflammation comparable to the diffuse interstitial type of inflammatory lesions of the parenchymatous organs in congenital syphilis.

The rarity of the occurrence of gummatous lesions in metasyphilis is too well known to be commented on other than to point out that in this respect there also exists a close analogy between metasyphilis and syphilis congenita praecox.

Most pathologists claim that in congenital syphilis the central nervous system is relatively rarely affected by the disease. Ranke's observations, however, are not in full accord with this view. He had an opportunity to subject to careful histologic examination twelve cases of syphilis in stillborn and newly born infants. He found in most of the cases evidences of a fibroplastic-infiltrative meningitis with numerous plasma and some mast cells. In a few cases the connective tissue proliferation was unusually well marked, and in two cases there were present necrobiotic foci with immature cellular elements. The meninges showed macrophages. In several cases the cerebral substance contained extensive adventitial infiltrations in the lymph sheaths consisting chiefly of plasma cells, and the walls of the blood vessels showed proliferating and degenerative processes. It is also noteworthy that the cerebral cortex as well as the white matter contained numerous cells resembling the *Stäbchen* cells of dementia paralytica. In addition to these more or less diffuse lesions there were also observed sharply limited encephalitic foci near the blood vessels. According to Ranke, the entire process in the brain resembled that of dementia paralytica.

As to organ localization of the pathologic process: Metasyphilis affects first of all the central nervous system, sparing all other organs except the aorta. It was Herxheimer who pointed out that there exist in congenital syphilis more noxae than in the acquired form to attract the spirochetes and thus influence the localization of the pathologic process. In a growing organism the various phenomena of growth and development demand a greater blood supply for proper functional activity of the organism. This fact may be of significance in determining in congenital syphilis the great intensity and more common localization of the disease in the liver and the relatively late appearance of the cutaneous lesions. This becomes evident when one bears in mind that the skin does not mature until late in the process of development, because it is not called on to function until the organism is ready to assume its extra-uterine existence. Of similar significance may also be the fact that in mesaortitis the nonspecific inflammatory cicatricial process in a definite location in the vessel wall represents, as in dementia paralytica, a primary tissue necrosis due to the parasite; the same may also be said of the lesions designated as miliary gummas, but which should better be called "miliary syphilomas" because they are not true gummas; true macroscopic gummas occur only in exceptional cases. Of no less significance in this connection is also the greater frequency of spirochetes in mesaortitis than in the tertiary stages of syphilis. The lesions in mesaortitis would seem to indicate that syphilis congenita praecox and metasyphilis have some common peculiarities, which occur in the latter not only in the brain but also in the aorta. These common peculiarities are attributed by Sträussler to the same type of a peculiar lack of

immunity common to both these conditions. In syphilis congenita praecox the defensive mechanisms of the organisms have not as yet come into existence, and in metasyphilis they have been lost in the course of the disease, although under certain conditions, such as proper antisyphilitic therapy, these mechanisms may have been regained.

According to Sträussler, the analogies pointed out thus far may be of some significance in explaining the pathogenesis of dementia paralytica. Two principal theories are generally invoked to explain the latter: 1. Willman's theory. Willman believed that dementia paralytica is due to a peculiar spirochete or to a peculiar reaction of the organism affected by the parasite; i. e., the organism is either constitutionally defective, or it has lost its power of resistance to the infection. 2. Jakob's theory. Jakob's formulation was that in dementia paralytica the general body reaction is poor and that in the absence of sufficient antibodies to inhibit the increase of spirochetes there results a malignant type of syphilis of the brain. Spielmeyer is opposed to this view because it assumes the nonspecific inflammation of the brain in dementia paralytica as evidence of malignancy of the process, although it is well known that nonspecific inflammation may occur in any syphilitic process in the early as well as the late stages of the disease. Spielmeyer also objected to Jakob's theory because it assumes the frequent occurrence of gummatous changes in dementia paralytica as evidence of an insufficient attempt of the tissues at a specific reaction which converts the syphilitic process into a benign one, whereas actually the occurrence of gummatous processes in dementia paralytica is unusually rare. Sträussler, while in general accord with Spielmeyer, believes, however, that the attempt to regard dementia paralytica as a malignant form of syphilis and to place it in juxtaposition to tertiary syphilis is not wholly without merit. Admitting that the occurrence of spirochetes in gummas is rare, Sträussler still believes that an organism capable of producing gummas also possesses sufficient reactional and defensive capacity to prevent a diffuse invasion and spreading of the spirochetes to the cerebral substance, with the formation of extensive degenerative changes of the type observed in the brain in dementia paralytica. There is no doubt that, biologically, as far as malignancy of the syphilitic process is concerned, the primary necroses in the brain are due to an undisturbed multiplication and accumulation of spirochetes. These facts lend further support to the analogy between congenital syphilis and metasyphilis.

Sträussler next calls attention to the frequent association of dementia paralytica with tertiary syphilis since the introduction of malarial therapy. He cites Kufs, who believed that in some cases of dementia paralytica malarial therapy affects the body so that it changes its mode of reaction, with the result that hitherto quiescent foci of spirochetes are stimulated to the production of specific tertiary syphilitic processes. Such a change in the organism can occur only with a simultaneous reactivation and increase in the protective and defensive mechanisms of the body which have become enfeebled during the stage of metasyphilis.

Finally it is pointed out that Dattner has shown by means of the cutaneous reactions of untreated patients with dementia paralytica to old tuberculin that most of them were anergic when the disease developed, whereas persons with dementia paralytica during the remission or stationary stage were not only allergic, but, in an unusually large number of cases even hyperergic. That neither the anergy nor the hyperergy can be attributed to the skin alone has been proved by Skalweit, who found that persons with dementia paralytica, in contrast to patients with cerebral syphilis, show at the beginning of the disease a leukopenia, whereas during a remission and following treatment a leukocytosis as an expression of increased resistance of the organism develops. Similar phenomena have been described by Benevenuti, who reported positive luetin reactions in patients with dementia paralytica during a remission, whereas at the onset of the disease their luetin reaction was negative, and by Alexandra Adler, who found that the previously rapid sedimentation of the red blood cells in dementia paralytica becomes normal after effective and successful therapy.

Sträussler believes that further researches in this and similar directions may lead ultimately to a better understanding of the pathogenesis of dementia paralytica.

KESCHNER, New York.

CONGENITAL BILATERAL PALSY OF THE ABDUCENS. WILLIAM H. PHILLIPS, JOSEPHINE K. DIRION and GRANT O. GRAVES, Arch. Ophth. **8**:355 (Sept.) 1932.

The authors present an interesting and unusual case, instructive from the standpoint of the opportunity for study of the histopathologic picture of a congenital bilateral abducens palsy associated with a so-called "status thymicolymphaticus." The case was that of a child, aged 5 years, showing bilateral palsy of the sixth nerve. Death occurred from the anesthetic during an operation on the muscles. The summary of the microscopic pathologic picture follows.

There were decided abnormalities in the nuclei of the sixth nerve, which were diminutive and had few cell nuclei. These diminutive sixth nuclei were about 1 mm. in diameter and were confined within an area bounded by the genu of their respective facial nerve. The genu on each side was also smaller than that in a normal brain, which is usually 1.7 mm. in diameter, according to Sabin. In addition, the nuclei were not well defined and not quite in their normal position. The nuclei were above the planes of the genu in their whole extent, not at and below the planes, as was found in a normal brain stem. There was no suggestion of a deviation from the normal in the shape of the genu, the cephalad embryonic shift of the diminutive sixth nucleus or the caudal and ventrolateral shift of the facial nucleus.

Descending from the region of the diminutive sixth nuclei were a few scattered fibers. None of these could be seen with the unaided eye from an inspection of the sections. The fibers were in strands of two or three, and extended only a short distance into the pons. No fibers were seen which reached from the sixth nucleus to the medial lemniscus; the usual extent was one fourth or one fifth of this distance. In some sections, the ends of these fibers did not seem to be pointing in a ventral direction, but turned to point laterally. These few fibers were directed as the sixth nerve would be if present and in normal proportions, that is, downward, ventrally and from just lateral to the genu. The possible termination of these fibers could not be determined by careful examination of the sections. In certain slides a few isolated triangular cells, similar to those of the nuclei, were found along the course of these fibers.

In the same sections through the sixth nucleus, there were found perhaps 50 nuclei, of which ten were triangular and deeply stained, as is characteristic of the normal sixth nucleus. The majority of sections through the sixth nucleus showed, however, a total of less than one half of the normal number of ganglion cells, the usual average being from eighty to one hundred triangular nuclei, in the center of the normal sixth nucleus. The largest number of these cells were faintly stained and contained round nuclei, which are seldom seen normally. The cells of the diminutive nucleus showed the ganglion cells to be densely packed in some of the sections, which is in marked contrast to the widely placed cells of the normal sixth nucleus. Other sections presented only a few cells of either type.

Pathologically, there appeared to be little evidence of fibrosis in the area near the sixth nucleus. The light area, however, above the location of the diminutive sixth nucleus was perhaps more richly supplied with blood vessels than is the corresponding area of the normal brain. This, nevertheless, does not suggest a definite pathologic change.

The surrounding nuclei seemed to be of normal size and of normal shape. Of the nuclei near the midline, for example, the twelfth nucleus was the usual distance from the genu of the facial nerve (about 1 mm.).

An accompanying abnormality was noticed in the immediate vicinity of the small sixth nucleus. There appeared to be an aberrant fiber tract just medial to the nucleus, which when traced caudad was found to be connected with the nucleus eminentia teres.

The fibers of the nucleus eminentia teres gathered together in bundles at first on their respective sides, but as they passed cephalad they united into a single median bundle. This bundle of fibers occupied a special median elevation in the floor of the fourth ventricle, not seen in the normal brain stem. In fact, the nucleus eminentia teres appeared abnormally long, extending as far cephalad as the beginning of the genu of the facial nerve. As the nucleus eminentia teres ended, the bilateral fiber bundles increased greatly in size. This fiber tract extended cephalad to the level of the superior limit of the genu of the facial nerve, where it again divided into multiple bilateral bundles.

This division of the aberrant fiber tract was associated with a small nucleus dorsolateral to it and dorsomedial to the asceunding fibers of the genu of the facial nerve. It may be that this small nucleus was an isolated part of the nucleus eminentia teres, for its position and the character of its cells were the same, although it might have been a detached part of the sixth nucleus, which, however, is less likely. There was a possible connection of the tract fibers with this nucleus, although the great majority, if not all, of them extended farther cephalad, disappearing at the level of the lateral shift of the medial longitudinal lemniscus. The fibers seemed to disappear laterally into the light areas immediately beneath the floor of the fourth ventricle at this place.

The complete absence, in the case reported, of both abducens nerves and their diminutive sixth nuclei and the presence of marked thymic hyperplasia, with its well known train of cardiac and vascular hypoplasias, lead one to the thought that perhaps bilateral congenital palsy of the external recti may be only another of the hypoplasias dependent on the thymic disturbance, rather than the result of an injury or hemorrhage sustained at birth. At any rate, it should make one careful in operating in these cases without careful investigation of the thymus beforehand.

SPAETH, Philadelphia.

THE SENSE OF SMELL IN THE NEUROSES AND PSYCHOSES. A. A. BRILL, Psychoanalyt. Quart. **1**:7 (April) 1932.

As an introduction to his subject, Brill points out that whereas animals, primitives and children still are markedly influenced and guided by the sense of smell, civilized mankind is gradually losing this sense, despite the fact that odors in the form of perfumes still continue to play a great part in the feminine life of even the most civilized people. He mentions the infrequency with which disturbances of smell are mentioned in modern psychiatric literature, despite the fact that Freud has indicated that the abandonment of smell-pleasure plays a part in the genesis of the neuroses, because of the erstwhile intimate association between the sex instinct and the sense of smell, and that with the advance of civilization, when the sense of smell fell more and more into desuetude, the sexual life of man, which was intimately connected with it, has perforce suffered in consequence. He then cites several cases: In the first, the patient had markedly impaired vision and hearing and congenitally absent smell and taste (only his tactile sensibility being intact); he had never experienced any sensation of hunger or sex desire, and as a consequence, being able to obtain erotic gratification only through tactile sensations, was a masochist of the flagellant type; this illustrates the dictum that normal sexual development must depend on the existence of an unimpaired sensorium. Perfect sight, hearing, taste, smell and touch will surely be more conducive to thorough and quicker appreciation of the love object.

In two cases of compulsion neurosis, halitosis was the outstanding complaint. This symptom could be traced to early pleasure in smell emanating from the mothers of the patients. One patient's sex potency depended entirely on the sense of smell; if a woman had any odor recalling his mother, the patient became impotent. Most of his outlet was of a peculiar auto-erotic nature. He rubbed his testicles and then smelled his hands, eventually attaining an ejaculation. Here the neurosis represented the negative of the perversion. Instead of getting partial or complete sex gratification through the sense of smell he was constantly afraid of affecting his environment through his malodorous breath, halitosis.

In two other cases of compulsion neurosis, with strong interest in the sense of smell, the patients were able to sublimate this interest after analysis by engaging in odorous occupations — one as a perfumer, the other as a cultivator of flowers.

Little has been written about the sense of smell in the psychoses, but whenever it is found as a symptom the patient usually conceives the odor as disagreeable, even if the odor happens to be sexually stimulating.

Another case illustrates that the rôle of the sense of smell may become particularly enhanced and sexualized when, as a result of an organic determinant, the person lacks the sense of sight. In this patient the sense of sight was congenitally absent, the sense of smell, which functions strongly only in childhood, not only continued but attained the same level as in animals and primitives. The patient became a necrophilic, his libido finding its greatest expression through the olfactory and gustatory senses. His greatest desire was not only to wallow in slimy carrion, but also to enjoy its odors and taste.

It is also well known that odors long buried in the unconscious may suddenly be brought to the surface by some association and in some cases may produce very disturbing effects. This effect may be very sudden, as in the case of a successful lawyer who suddenly sexually assaulted an elderly charwoman, being aroused to do so because of the odor of stuffy humid air, sour perspiration, dirty water and the wet floor. Occasionally, sublimatory activities or vocations are direct descendants of the sense of smell. Brill cites certain cases to show that such patients unconsciously endeavor either to mask or to replace the unpleasant odors of their mother cathexis by perfumes, flowers and pleasant scents and sights, but that they all still adhere to the odoriferous mother.

The first conclusion is that, although the sense of smell has been almost completely superseded and displaced by sight and touch, it surely continues to play a part, albeit an unconscious part, in the mental life of man. Nevertheless, there is no question that the sense of smell has been subjected to deeper repression than any other sense. Although the sense of smell unconsciously plays the same part in all love manifestations of civilized humanity as it does consciously among animals and primitives and many non-European races, it has been subjected to greater repression than all the other sense organs. This has probably come about because the sense of smell fell into disuse at about the time that the first sex taboos were established. But as these taboos were primarily of an incestuous nature and smell was still an active sex function, primitive man was under special stress to curb this sense. The hypnotic sex-attractive odor given off by the female in "heat" must have been one of man's greatest temptations to violate the incest taboo. Olfactory pleasure has been subjected to such deep repression for so long that even when the repression fails and allows the odors to appear in consciousness they do so without any retrogression of the repression, in the form of symptoms. The prognosis of schizophrenias with olfactory hallucinations is invariably bad.

Brill concludes by quoting from Maugham that the reason for the real democracy of the despotic East and the real despotism and class distinctions of the democratic West lies in the fact that in the former there is little or no distinction on the basis of cleanliness and odorlessness, while in the latter such a distinction is evident. The cesspool is more necessary to democracy than parliamentary institutions. The invention of the "sanitary convenience" has destroyed the sense of equality in men. It is responsible for class hatred much more than is the monopoly of capital in the hands of a few.

PEARSON, Philadelphia.

REPORT OF THE COMMITTEE ON MENTAL DEFICIENCY. PRESENTED TO THE ANNUAL REPRESENTATIVE MEETING OF THE BRITISH MEDICAL ASSOCIATION, JULY, 1932.

The terms "idiot," "imbecile" and "feeble-minded," as defined by the British Mental Deficiency Act, are acceptable to the Committee, although the expression "moral defective" meets with less universal approval. Excluded from the category of moral defectives are delinquents, whose difficulties are due in large part to social and emotional maladjustments, and persons suffering from epilepsy, syphilis,

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encephalitis, injury to the head, chorea and the neuroses, whose inadequacies can be explained on the basis of the disease picture. However, "moral defect" is properly included within the broad scope of mental deficiency on the theory that it is due to a failure of the subject to develop the capacity for wisdom, judgment and control, based possibly on actual cerebral disease. Morally defective persons, because of their normal intelligence and persistent tendency toward delinquency, are not amenable to ordinary institutional care and need special accommodations.

The committee is satisfied that heredity is an influence in the determination of mental deficiency, but is unable to state in what proporation of cases it constitutes a significant factor. It seems likely that there are several congenital bases, the trend being transmitted as a multiple recessive complex of traits. The x-rays and radium can certainly produce inheritable mutations in the genes, but the influence of chemical toxins is less definite. There is no reason to believe that tuberculosis, lead poisoning or intoxication due to mercury in either or both parents is likely to be followed by defective offspring. Syphilis may certainly be associated with mental defect, but the relationship is not so simple and direct as is commonly assumed. The coexistence of clinical evidence of congenital syphilis, a positive Wassermann reaction, and mental deficiency constitutes no absolute proof that the latter is due to the former. In a definite but small number of cases, alcoholism plays a part. The claim that, in any particular case, injury at birth was the precipitating factor must be examined with critical care, for the sequence does occur but is rare. Postnatal trauma is an even more infrequent etiologic factor. Infections, especially encephalitis, seem in many instances to be the more likely agencies.

The committee warns against the stigmatizing of a child as mentally defective because of inadequate success at school, pointing out that failure at school is not incompatible with excellent social adjustment. It recommends the utilization of tests in achievement and aptitude as supplementary to the conventional psychometric examination before a diagnosis is made.

For the care of the mentally defective, the committee much prefers the large colony to the small home or the hospital for mental patients. The large colony possesses many advantages: employment can be organized on a scale sufficiently wide to assure suitable work for every inmate; patients of higher grade mentality can be segregated and specially trained; economy is effected by reduction in the overhead charges, and life can more closely approximate normal communal intercourse.

The only method of prevention discussed is sterilization; as to the desirability of this, the committee is uncertain, feeling that if this operation were universally applied to all of the committable feebleminded, no reduction in the incidence of mental deficiency would be noted. This is because of the large numbers of unreachable carriers who could transmit the defect. Even if those patients whose chief social danger is propagation would be sterilized, the advantages would be uncertain because of the sexual promiscuity and consequent spread of venereal disease to which such a practice would lead.

The commitee is inclined to look for relative disproportion in the intra-uterine development of neuroblasts and spongioblasts as a common pathologic base for many cases of mental deficiency. Certainly cases of hydrocephalus and microcephalus can be explained in this manner. The controversy between heredity and environment is resolved by the committee in the phrase: "Heredity furnishes the material, environment shapes and uses it; heredity is the mechanism, environment, the stimulus which sets it in action; heredity fixes the possibilities of development, environment determines which of these possibilities shall, and how far they shall, become realities." Studies in comparative anatomy and anthropology seem to suggest that it is to a defect in the numbers rather than in the structure of the brain cells that one should look for the factors that underlie mental deficiency.

Many of the maladjustments suffered by the feebleminded are explicable more on the basis of their environmental difficulties than on the theory of their fundamental inadequacy. The high grade moron, who adequately performs a humble and necessary task, is an effective unit in the social machinery and should not be

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patronized as "unfit." Certainly, antisocial conduct is not even a common, much less a necessary, characteristic of the mentally deficient. These people need to be protected from society as much as society needs to be protected from them. The committee considers that this rapidly growing problem is generally overstressed, for it has found no evidence that procreation is excessive among the feebleminded, or that the number of children in mentally deficient families who reach adolescence exceeds the number in normal families.

The report is concluded with several appendixes dealing with the problem of marriage, illegitimacy and community care of the mentally deficient. Emphasis is laid on the ultimate aim of the recommended program—the socialization of the mental defective.

DAVIDSON, Newark, N. J.

SIZE AND SHAPE OF OCULAR IMAGES: II. CLINICAL SIGNIFICANCE. ELMER H. CARLETON and LEO F. MADIGAN, Arch. Ophth. **7**:720 (May) 1932.

This is the second of a series of articles on "The Size and Shape of Ocular Images" from the Department of Research in Physiological Optics, Dartmouth Medical School. The entire study, as represented in the three articles mentioned, is the joint work of Ames, Gliddon, Ogle, Carleton and Madigan.

The patients showing ocular symptoms and those showing general symptoms of ocular origin who obtained no relief from accepted therapy form a large group, apparently, in the practice of every ophthalmologist. In a certain percentage of these cases a diagnosis of ocular neurosis can easily and, perhaps, correctly be made. In many cases, however, such a diagnosis would not meet the situation. For this reason, the possible existence of some physiologic factor, heretofore overlooked, seemed to justify a careful investigation to account for some of these disturbing symptoms. Surely a diagnosis of neurosis should not be made until it is definitely determined that there is no physiologic cause for the disturbance.

The patients used for this experiment were, in general, those who had not been relieved by previous refractions. There were three general groups. One group did not show strictly ocular symptoms. This group was examined for ocular defects, and that factor as the chief or contributing cause was considered. The second group consisted of patients whose symptoms were manifestly ocular. The third groups included patients who had a combination of ocular and nonocular symptoms, and who showed signs of stress and discomfort somewhere along the sympathetic tract.

In this experiment, the patient's refractive condition was first measured; i. e., with binocular fixation at 20 feet (6 meters) the point in space that is conjugate to each retina was determined. In this way it was determined whether the images were equal in size, or whether one image was larger over all than the other or merely larger in one meridian than in the corresponding meridian of the other eye. The errors in refraction were then corrected by trial lenses. Any difference in size in the retinal image was equalized by the lenses described in a previous paper. When the refraction was balanced and the size of the ocular images equalized, the combined refractive and size lenses before the eyes were considered to be the correction necessary for that particular distance. The same procedure was completed for near vision at 16 inches (40.6 cm.).

From this point various factors were studied. They included: suppression, as a certain number of cases showed that a partial or local suppression had taken place at the fovea; different size correction for distant vision and near vision; size amplitude; change of slip with size and the displacement present with those changes in relationship to size, and the effect of lenses of incorrect size. Prisms were not used in any of the cases, because of the distortion of the ocular images produced by them.

The statistical analysis of the cases covers the age, refraction and phoria of the patients, their symptoms, the type and amount of size differences, and the clinical effect of correction of size difference. A group of cases are then outlined in detail and the article closes with a summary and conclusions, the following of which are considered to be of importance.

1. Difference in the size of the ocular images of the two eyes may exist without causing discomfort, but in such cases the differences were less than when there was discomfort.

2. Differences in the size of ocular images associated with ocular discomfort were found in persons of all ages with emmetropia, all types of refractive errors and orthophoria as well as with all types of heterophoria.

3. The symptoms associated with the difference in the size of the ocular images were of two types: those apparently involving the sympathetic nervous system and those of an ocular type.

4. Correction of size differences apparently brought complete relief in about 20 per cent of the cases, partial relief in 60 per cent and no relief in 20 per cent.

The evidence presented that the existence of this new type of physiologic defect may be the cause of various ocular troubles would seem to show that the ordinary methods of measurement do not disclose all the abnormal physiologic conditions that may exist and that, at least in all doubtful cases, too definite conclusions should not be formed until measurements have been taken to determine the relative size and shape of the ocular images. SPAETH, Philadelphia.

IS THERE A MENTAL HYGIENE? FRANKWOOD E. WILLIAMS, Psychoanalyt. Quart. **1**:113 (April) 1932.

The author comments on the extreme difficulty of defining the term mental hygiene. He points out that although in the past twenty-two years the world has changed considerably in manner of thinking and approach to and method of handling social problems, the movement of mental hygiene has not changed. It remains today essentially what it was in the beginning, a philanthropic and artificial movement. The present point of view in the field of mental hygiene throughout the world is clearly revealed in the *Proceedings of the First International Congress on Mental Hygiene,* in which is reported a series of special meetings known as "World View of Mental Hygiene," at which the chief representatives of fifty-three countries, psychiatrists in each instance, discussed the status of mental hygiene in their own countries. With scarcely an exception, the statements have to do with the efforts being made to increase the facilities and to improve the care and treatment of the insane, feebleminded and epileptic. There is an occasional mention of diagnostic work in connection with delinquency and of the extension of diagnostic and therapeutic service into the community. The social value of this work is not to be denied. But is it mental hygiene?

On the other hand, one observes growing spontaneously outside of organized mental hygiene a very considerable feeling of the need of mental hygiene, although it may not be known as such. Persons in the fields of sociology, education, law, social work, international relations and religion, working independently of each other, each dealing with the material, methods and technics of his own group, have come by reason of the logic of their own material and experience to the same point of frustration. As from these various directions this point of frustration is reached, an acute need arises, a need which can be summed up in the question, What are the motivations of human conduct? This is a question relating to mental hygiene, and its present rise is a challenge to mental hygiene. The term "mental hygiene" implies knowledge of, if not an answer to, this question.

Challenged by this situation, those of us who have dared to call ourselves mental hygienists must face the question, Is there a mental hygiene? Can there at this time be a mental hygiene? From what source or sources can this mental hygiene develop? If all we have to offer is an ameliorative program in social psychiatry or such advice as "Avoid syphilis, and you will avoid syphilitic psychoses," or "Avoid alcohol, and you will avoid alcoholic psychoses," or "Don't worry," "Keep smiling," "Know thyself," "Face the facts" or "Face reality" or common principles of physical hygiene, such as adequate sleep, recreation and guarding oneself against too great physical and nervous strain and the like, then we must cease calling ourselves mental hygienists, face the facts that we have no

mental hygiene and, if we have the courage and can free ourselves from our own presumptions, attempt in a humble spirit to discover what we have only been talking about.

From what source or sources will a real mental hygiene come? Neurology has contributed nothing to knowledge of the neuroses; psychiatry has contributed nothing to knowledge of the functional psychoses. Neurology and psychiatry need no justification. Their value is not to be judged by their contribution or lack of it to mental hygiene. Perhaps a mental hygiene is not possible at the present time. If this is the case, then it would be well to admit it, for that would in itself be a beginning.

The basic question with which psychiatrists, and particularly those interested in mental hygiene, start is, What are the causes of mental and nervous disease? This question has been repeatedly raised during the twenty-two years of organized mental hygiene, until it has almost become a ritual and like a ritual has led to nothing except repetition; it has not even a start. From the point of view of mental hygiene perhaps we may get further by beginning with what would seem to be a more fundamental question, What are the motivations of human conduct?

To answer this question we must begin at some strategic point; that point obviously is infancy. Although aware of the unity of the infant, mental hygiene will focus its attention on the developing psyche, attentive to but leaving to others other types of investigation. Because it is at hand and has proved productive and useful, infantile sexuality is the natural point of departure, and for this study psychoanalysis is the natural working hypothesis of choice.

PEARSON, Philadelphia.

THE COSTOPECTORAL REFLEX: ITS SEMIOLOGIC VALUE IN MEDULLARY COMPRESSIONS. G. NOTO, Riv. di pat. nerv. **40**:477 (Sept.-Oct.) 1932.

Noto describes two cases of compression of the spinal cord due to extramedullary tumor at the level of the third and fourth dorsal vertebrae. In both the costopectoral reflex, which was first described by Noto (*Boll. d. Soc. ital. di biol. sper.* **5**:508, 1930), was present. To elicit the reflex the body of the seventh rib is percussed with a hammer in a zone between the anterior and middle axillary line. This provokes a contraction of the inferior bundles of the large pectoral muscle. Contraction of this muscle is sometimes better felt by applying the hand over the margin of the anterior wall of the axilla. At times a slight movement of adduction of the arm is noticeable. In cases of extreme activity of the reflex, the contraction of the pectoralis major is accompanied by a simultaneous contraction of the superior muscles of the abdominal wall. No contraction of the contralateral pectoral muscle takes place. The patient must lie on his back in a state of complete relaxation, while the arm is kept slightly abducted from the trunk. In some cases the reflex is better put in evidence if the arm is kept extended upward beside the head and the forearm is flexed around the head. Percussion must be strong enough to stimulate the underlying bone tissue. This, combined with the fact that in the stimulated area there are no insertions of the pectoralis major, establishes that the reflex is exclusively periosteal and does not depend on an idiomuscular contraction of the muscle. The center for the reflex is located at the level of the eighth cervical and the first dorsal vertebrae; the centrifugal pathway is the grand pectoral nerve; the centripetal pathway is the seventh intercostal nerve, which enters the spinal cord at the level of the seventh dorsal segment. From that point upward the reflex path becomes intramedullary, and in order to reach its center travels from the seventh to the first dorsal vertebra or to the eighth cervical segment in the posterior columns. It is because of this intramedullary course of the afferent pathway that the costopectoral reflex has value.

Any pathologic process that interrupts the conductivity of the spinal cord, or at least of the posterior columns, at any level between the first and the seventh dorsal vertebrae will cause the disappearance of the reflex. As these levels contain no other centers of clinical spinal reflectivity, this new costopectoral reflex is the only available method for establishing its integrity. It is true that there are

other reflexes which result in a contraction of the pectoralis major, for instance, the tendinous reflex of the pectoralis major, the scapulohumoral reflex of Bechterew and the mediosternal reflex, all of which have centers at the level of from the eighth cervical to the first dorsal verebrae. But the afferent pathway is different, as the reflex is elicited through stimulation of areas higher than the one indicated for the costopectoral reflex, and hence the semiologic value is different.

To establish particular value for this reflex, the author does not accept Hoffmann's explanation of the mechanism of the deep reflexes. Hoffmann believed that percussion of the deep structures, is transmitted to the muscle and that the reflex takes place through afferent fibers which through the muscles reach directly the center of the reflex. In one of the two cases illustrated by the author there was integrity of the center of the reflex, and yet the reflex was abolished, a fact which the author correlates with a lesion of the intramedullary afferent pathway between the seventh dorsal segment and the center for the reflex. In another case the author reported an increase of idiomuscular excitability though the costopectoral reflex was absent, thus establishing the independence of the reflex from the excitability of the muscle itself.

FERRARO, New York.

THEORETICAL DEDUCTIONS FROM THE PHYSIOLOGY OF THE CEREBRAL CORTEX. D. DENNY-BROWN, J. Neurol. & Psychopath. **13**:52 (July) 1932.

This article is a critical discussion of Pavlov's work on conditioned reflexes and should be read by all who are interested in the physiology of the cerebrum. The author objects to Pavlov's use of the terms reflex and inhibition. Reflex connotes to Denny-Brown inflexibility of response, a necessary consequence of anatomically prearranged fixed nerve pathways, whereas conditioned reflexes are characterized by flexibility of response and habituation to stimuli. By using the term conditioned response he avoids calling all too many and varied reactions of the dog reflexes. In establishing a conditioned response there is an essential time element between the signal and response-exciting agent, and a fulfilment factor or satisfaction of a proprioceptive sensation (hunger), a factor not adequately discussed by Pavlov.

Inhibition as applied to spinal reflexes is an active negative process, similar but opposite in its effects to excitation. Pavlov tends to apply the term to states in which a conditioned response fails to occur, which may be mere passive absence of response. The inhibition produced by a new or unusual stimulus might be explained by a transfer of attention, which attention facilitates all conditioned responses. "May it not be that the process which underlies cerebral focus or concentration is, for instance, a reflex link which, by virtue of a strangeness or newness in the environment, is added to otherwise incomplete nervous circuits to allow the easier passage of impulses over the pathways connected to the temporarily most appropriate receptor?" The inhibition of other conditioned reflexes in the time interval of a trace reflex may be related to the functioning of the cortex in estimating time. Slight external distraction of the attention is also adequate to explain the phenomenon of "disinhibition," i. e., the return of an original positive response after a negative conditioned response has been grafted on it.

A conditioned response at first is generalized to similar kinds of stimuli, the properties of the stimuli in differentiation thereby being given signal value; i. e., new reflexes are established in response to the specific properties of the stimuli. Pavlov explains differentiation by the spread of the effective influence of points in the analyzer, but the author would prefer to do this on the basis of facilitation provided by the attentive process. Reaction to difference reveals the true analytic function of the cortex.

In regard to functional localization, Pavlov found by ablation experiments that only the finer discriminations in any range of sensation require the area of the cerebral cortex commonly accepted as the specific area for that sensation. The author believes that the present accepted functional localization in the cortex is the reflection of a relative concentration of like functional elements which are diffusely spread.

Sleep is explained as "the complete suppression or disappearance of the process of attention, leaving all cortical processes subliminal."

The author prefers to view cortical function as a pattern, the details of which are not essential, that is composed of manifold sensory and proprioceptive threads, rather than as a functionating chain or single pathway active at the moment.

SPERLING, Philadelphia.

THE FUNCTIONS OF THE OLFACTORY PARTS OF THE CEREBRAL CORTEX. C. JUDSON HERRICK, Proc. Nat. Acad. Sc. **19**:7 (Jan.) 1933.

In reptiles the entire pallial field is dominated by the olfactory system, but the dorsal cortical area receives many more nonolfactory fibers than do the corresponding areas of amphibians. In the lowest mammals this olfactory part of the pallium attains considerable development, and from this point onward in phylogeny develops very little further. The neopallium on the other hand, which is very small in the lower mammals, grows rapidly, attaining enormous size and differentiation in the higher animals of the mammalian group. The conservatism of the olfactory areas is presumably associated with the relative decrease in the importance of the sense of smell in the course of mammalian evolution.

Characteristic of olfactory sensation, even in the animals in which it is highly developed, is the fact that it is never an efficient localizing sense. Odors may be discriminated, but they cannot be accurately located without the use of other senses. Accordingly, the olfactory pallium is anatomically associated with all other parts of the cortex and many parts of the subcortex. In reptiles and in amphibia the cortex is a general organ with no well defined nuclei or centers, and it is only in the mammal that one finds cortical localization and functional segregation. Yet none of these areas, even in animals with highly developed olfactory mechanisms, can be said to be truly olfactory centers in a sense comparable to centers for vision or hearing. This is further evidence of the associative rather than initiative or interpretative function of the olfactory pallium.

Ablation experiments in lower animals almost always leave the olfactory areas intact. Since it is precisely these areas which are capable of integrative and nonspecific function, any conclusions as to the functions of the ablated areas must be considered incomplete. Herrick suggests experiments on mammals in whom the neopallium is left intact and the olfactory parts of the cortex are removed. Evidence of the potentialities of the olfactory zones is afforded by anosmic animals such as the dolphin, which preserve well a differentiated hippocampal cortex in spite of the absence of a peripheral apparatus for smell.

The inferences Herrick draws from these considerations are: 1. In exteroceptive adjustments, the olfactory sense, lacking localization of its own, cooperates with other senses in various ways, activating and sensitizing the central nervous system as a whole. 2. Differentiation of tissue within the pallial field began in phylogeny with the penetration into this part of the primitive olfactory area of various systems of nonolfactory projection fibers, which results in more efficient adjustment to all situations containing olfactory excitation. 3. At all stages of cortical elaboration an important function of the olfactory cortex, in addition to participation in cortical associations, is to serve as a nonspecific activator for all cortical activities.

DAVIDSON, Newark, N. J.

CLINICAL CHARACTERISTICS, HISTOPATHOLOGY AND INHERITANCE FACTOR OF HIPPEL-LINDAU'S DISEASE. H. KUFS, Ztschr. f. d. ges. Neurol. u. Psychiat. **138**: 414, 1932.

The occurrence of a cerebellar cyst with angiomatosis retinae and cyst formations in other parts of the body has been well described by Lindau. Several cases of this type have been recorded. Kufs reports a recent case with a cyst in the cerebellum, in which the ophthalmoscope showed no evidence of Hippel's disease (angiomatosis retinae). This does not exclude its presence, however, since Lindau reported a case in which no evidence of angiomatosis retinae was found

on ophthalmoscopic examination but in which careful microscopic study revealed a minute angioma. The solid tumor in Kuf's case occupied almost the entire area of the tumor, only a small part being cystic. Its structure was typically that of a hemangioblastoma.

The hereditary tendency in Hippel's disease has long been known. Wood and Collins (1892) reported angiomatosis retinae in two members of a family. Griffith and Ormond reported it in both eyes in a woman, aged 22, and in one eye of her sister, aged 14. Fenkel reported bilateral angiomatosis retinae in a brother and a sister. Brandt described it in a father and son. Tresling reported angiomatosis retinae in two brothers; these cases were studied further by Rochat, who reported that in one of the brothers signs of tumor of the brain later developed and an operation was performed. Angiomatosis retinae and cerebellar tumor developed in a third brother. A small angioma in one eye developed in a son of a healthy sister. The family is of interest in showing the development of optic and cerebral signs simultaneously and also because it demonstrates the inheritance of the disease through a healthy member of another generation. Lundogaard reported Hippel's disease and cerebellar angioma in a patient; two brothers and two daughters of one of the brothers had tumors of the brain. Rochat described a family in which the disease was followed through three generations; the grandmother died at the age of 28 of a cerebellar tumor; she had seven children, two of whom died in youth, three having suffered from a cerebellar angioma; the healthy daughter had eight children, of whom 1 showed retinal angiomatosis in one eye. Moller traced Hippel-Lindau disease through three generations. The father died of a tumor of the brain and a son died of a tumor of the cerebellum and medulla oblongata, with angioma of the retinas. A second son died of a nervous disorder, and a third died of Lindau's disease. Two daughters in the third generation suffered from the disease. Kufs believes that Hippel-Lindau disease is an inherited dominant characteristic but that it may be inherited through apparently healthy members of a family, who act as conductors of the disease.

ALPERS, Philadelphia.

CLINICAL CONVULSIONS. FOSTER KENNEDY, Am. J. Psychiat. **11**:601 (Jan.) 1932.

Epileptic seizures constitute no disease unit, but must be considered rather the expression of an instability of nerve centers of diverse origin operating through a common connecting mechanism, probably the cardiovascular system. The tonic phase may best be understood as a sudden cutting out of the highest level, so that the cortex permits the lower neuronic strata to pour downward an ungoverned stream of tonic postural impulses. The gradual return of cortical influences, incoordinate and unvolitional, produces the clonic phase. Of similar pattern, but of different intensity, are all epileptic phenomena from the seizure of grand mal to internally produced periodic emotional storms. The olfactory, auditory and visual hallucinations and illusions, which often precede, accompany or replace epileptic seizures, are explicable on a phylogenetic basis. Each person in his ascent from embryonic life traverses the stages through which the race has passed, and these sensory apparitions may be considered involuntary resuscitations of early and infantile memory patterns.

Of all extrinsic factors that render a brain unstable, trauma is one of the the commonest. In 4 per cent of the cases of unstable brain occurring during the war, convulsions followed injuries to the skull and the brain; a dose of standardized convulsant too small to explode an intact brain, will explode an injured one. The nature of the seizure depends on the location of the sensitive area; petit mal is associated with disorder of the frontal lobe, myoclonic attacks with striatal implication, cataplexies with thalamic involvement and hallucinations with pathologic conditions of the appropriate sensory cortices, while the grand mal is to be considered a sudden cutting out of cortical totality.

Many patients suffering from periodical convulsive disorders present no evidence of external or intrinsic cerebral trauma; in some of these an unlooked

for trauma at birth may prove to be the predisposing agency. Bloody spinal fluid has been found in 11 per cent of 100 cases of apparently normal delivery, while in delivery with forceps, by breech and in prolonged labors the percentage of cerebrospinal hemorrhage is even higher. This suggests an approach to preventive medicine in the field of epilepsy requiring the cooperation of the obstetrician. Once the injury has been accomplished, efforts must be directed, first, toward raising the threshold of neural explosion, and second toward abolishing all possible detonating stimuli. Starvation, removal of infectious foci and the ketogenic diet are directed toward the latter goal, while the use of sedatives, notably phenobarbital, is at the time the best available method of raising the neural threshold.

DAVIDSON, Newark, N. J.

CLINICAL AND PHARMACODYNAMIC INVESTIGATIONS OF THE CEREBROSPINAL FLUID PRESSURE BY MEANS OF SERIAL MEASUREMENTS IN MENTALLY DISEASED PERSONS. K. HAUG, Arch. f. Psychiat. **97**:185 (July) 1932.

Measurements of the cerebrospinal fluid pressure are of value only if repeated over a period of time and under standard conditions, because otherwise some mental or physical disturbance may temporarily alter the pressure. The author undertook a series of experiments in which the patient was placed under certain standard conditions; the estimations taken every four seconds were recorded for anywhere from one to one and one-half hours. Twenty-two patients were used, with a total of thirty-five sessions, some of the patients being subjected to more than one session. The estimations were made: (1) under normal conditions; (2) under the influence of various stimuli, such as psychic, physical and pharmacologic, and (3) during normal sleep and genuine epileptic and pyknoleptic attacks. The psychic stimuli were those of pleasure, pain, fright and anxiety; the physical were pain, cold and warmth; the pharmacologic were epinephrine, histamine, caffeine, atropine, pilocarpine and others. Injections of hypotonic and hypertonic solutions were also given. The subjects were patients with feeblemindedness, epilepsy, psychopathic states, schizophrenia, senility, manic-depressive psychosis, paralysis agitans, multiple sclerosis and pyknolepsy.

The results were: Under normal conditions, that is, without artificial stimuli, two types were established: 1. Unimaginative, stuporous and emotionally deteriorated patients showed a fairly even curve, with very slight oscillations. 2. Contrary to this, patients with increased emotional tone and lability of affect showed marked variations in the curve, with up and down strokes of pronounced amplitudes. Acute psychic excitements, as well as the psychic stimulations mentioned, produced marked variations and up and down strokes in the curve. This was especially pronounced in cases belonging in the second group. In normal sleep and in pyknoleptic attacks there was a pronounced lowering of the pressure, but in epileptic attacks no definite change was found. Epinephrine, histamine and amyl nitrite caused consistent increases of pressure even though their effects on the blood pressure were not the same. Caffeine tended to produce a moderate fall in pressure. A pituitary solution had no effect. Afenil caused a slight increase in pressure. Atropine caused a moderate lowering of pressure, whereas pilocarpine caused a moderate increase. Most of the hypertonic and hypotonic solutions produced no appreciable change in the pressure. In only one case did distilled water cause an increase of pressure, and 20 per cent saline solution a decrease.

MALAMUD, Iowa City.

PATHOLOGIC ANATOMY OF THE CEREBELLUM. HANS JOACHIM SCHERER, Ztschr. f. d. ges. Neurol. u. Psychiat. **139**:337, 1932.

The object of this article was to report the changes in the white matter and nuclei of the cerebellum in various diseases. Changes in the white matter may be diffuse or focal. Among the former is senile retrograde change with an intact cerebellar cortex. In such cases there is a fibrous gliosis which is probably physiologic; it was found in all such cases studied by Scherer in patients of

from 65 to 85 years of age. The focal changes are more marked. They are the result of circulatory changes in arteriosclerosis, syphilis and embolic processes. Only once in Scherer's observations was a fresh softening found. Glial scars were frequent, and cysts and old softenings were rare. In one case there was a softening with a subsequent massive hemorrhage. Bohne has recently opened the question of cerebellar apoplexy; he thinks that the cerebellum is a site of predilection for hemorrhage, just as are the putamen and the pons. Scherer states that the cerebellum is not so rarely a seat of hemorrhage as is thought. Spielmeyer has shown that the cerebellar white matter is a site of hyaline changes in the vessels just as is the basal ganglion region; this may explain the tendency to hemorrhage; furthermore, there is a tendency for pseudo-calcium to be deposited in their walls. Functional vascular crises occur frequently in the cerebellar white matter. Inflammatory reactions are common but rarely affect the cortex; they are found in poliomyelitis and in encephalitis. Isolated perivascular infiltrations are found in the white matter in cerebral syphilis and congenital syphilis, less often in dementia paralytica and frequently in arteriosclerosis around hyalinized vessels.

As for the changes in the cerebellar nuclei, atonal chromatolysis is found in the dentate nuclei in pellagra, pernicious anemia, chronic encephalitis, some forms of epilepsy, arteriosclerosis, multiple sclerosis, catatonia, Lissauer's paralysis and carbon monoxide poisoning. Cell shrinkage is extremely common in the dentatum. Marked changes in the cells are rare; on the other hand, homogeneous changes are common. The other nuclei of the cerebellum show changes similar to those observed in the dentatum.

The changes observed in the cerebellar cortex (discussed in another article) are attributed to circulatory, toxic and inflammatory factors. Those in the cerebellar nuclei have another factor, bound up intimately as they are with the Purkinje cells. Changes in the latter system must be ruled out, therefore, in interpreting these findings. Toxic processes play a very slight rôle in changes in the dentatum.

ALPERS, Philadelphia.

THE DEMENTIA PRECOX CHILD. WINIFRED RICHMOND, Am. J. Psychiat. **11**: 1153 (May) 1932.

Psychiatrists are generally reluctant to make a diagnosis of psychosis in childhood, and except for instances of frank feeblemindedness or occasional juvenile dementia paralytica have attached the vague label of "psychopathic" or "inferior" personality to children presenting certain peculiarities of behavior. The author, however, is inclined to think that many of these children have true cases of schizophrenia. Such children constitute behavior problems in school and at work; they are incorrigible and cruel, unenergetic and solitary. They flee from reality, and literally run away from the responsibilities of school, home and industry. During the first ten years of their lives they are of normal intelligence (as determined by the usual psychometric studies), although they may appear at first glance to be mentally defective. In view of their normal intelligence and of the lateness of the deterioration, these children cannot properly be classed as "potentially feeble-minded."

Two cases are cited: The first was that of a sailor, aged 23, who was transferred from the naval base because of incompetence; he had never played with other children, preferring to invent queer games of his own. He complained that his mates teased him, and when confined in the guard house for an infraction of the rules would sing and shout of the punishment of God being visited on his tormentors. Although his mental age with the Binet test was only 9¾ years, his actual intelligence was certainly much higher than this would suggest. The second case was that of a boy, aged 15 years, with a mental age of 12, who gave a similar history during childhood of queerness, solitude, cruelty and failure to make domestic, scholastic or vocational adjustment.

A survey of one hundred and fifty admissions to St. Elizabeth's Hospital showed twenty-eight who were probably and fourteen who were almost certainly of this

type. It is the author's thesis that intensive study of children with the psychologic and psychiatric tools now available is accumulating evidence to suggest that a deteriorating process, closely comparable with dementia praecox, is at work from an early age, operating in children whose intelligence develops normally at first. A more careful study, from both psychiatric and psychologic approaches, of the patients in the child guidance clinic will undoubtedly reveal that many of them are of this type.

DAVIDSON, Newark, N. J.

CATALEPTIC SLEEP. H. CLAUDE, H. BARUK and R. PORAK, Encéphale **27**:665 (Sept.-Oct.) 1932.

Most discussions of voluntary action and volition center around concepts which are more or less metaphysical. The problem needs more study from the standpoint of physiology. Investigations of epidemic encephalitis as well as of catalepsy point out certain characteristics: slowness of beginning motions, prolongation of action and difficulty in cessation from action. There is an absence of spontaneous and adaptable activity. Yet cataleptic attacks may terminate under the influence of noise, psychic impressions and other fortuitous stimuli—stimuli which apparently act on some psychomotor cerebral center.

Jarkowski has insisted that volitional movement is tied up with an idea (cortex) on the one hand, and an affective phenomenon (basal ganglia) on the other. Studies by the writers do not corroborate these theories. Is it proved that volitional activity is tied up with the representation of an idea? It is not sufficient that a voluntary act be preceded by a corresponding idea; there must be concentration on the act. This is the essence of William James's contention that the effort of attention is the essential element of the will. This means that the important thing is a capacity for work—for synthesis—within the brain. Nor do the authors regard Jarkowski's affective factor, proto-energy, as fundamental. Cases of dementia praecox demonstrate that the trouble is not absence of proto-energy, but rather a complex disturbance of control and coordination. The cataleptic patient, when deprived of his volitional capacity or motor initiative, feels a particular impression of numbness or torpor, and at the same time experiences an undisciplined train of ideas. He cannot stop this flow of ideas, and all coordinating activity is hindered. He feels as though he is a spectator watching the unfolding of psychologic phenomena which he cannot direct or control. It is the suspension of the functions of synthesis and the appearance of an incoercible, automatic unfolding of phenomena that give the impression of suspended volition. Cases of intoxication offer evidence of this. In lower vertebrates there is no true motor initiative; the motor regimen consists of a "spontaneous akinesia, intercepted by impulsive and oft repeated reactions to excitations." Higher animals, on the other hand, are endowed with true motor initiative, "genuinely spontaneous, pliant, and adaptable." There is thus a parallelism of motor initiative and development of the neocortex.

ANDERSON, Los Angeles.

WRITERS' CRAMP. COSTEDOAT and AUJALEU, Paris méd. **22**:445 (Nov. 26) 1932.

Costedoat and Aujaleu take issue with Dejerine's sweeping statement of two decades ago that in writers' cramp only psychic causes need be considered. They are inclined rather to agree with Souques, who compared it to the movements of spasmodic torticollis and of chorea. Before even the present scanty knowledge of the physiology of the basal ganglia was available, one was prone to consider paralysis agitans and the various choreas as "functional" disorders. Writers' cramp may be in a similar category; cases of this occupational spasm in patients suffering at the same time from organic diseases of motility have occurred with more than coincidental frequency. Writers' cramp has been reported in conjunction with wry neck (Pierre Marie, 1922), with cervical spondylitis (Barré, 1924), with trauma of the cervical spine (Draganesco, 1924), with paralysis agitans (Lévy, 1921), with Huntington's chorea (Alajouanine, 1928), with cerebrospinal syphilis (Juster, 1928), with dementia paralytica (Paulian, 1928) and with injury to the left frontal lobe of the cerebrum (Cornil, 1929).

Costedoat and Aujaleu postulate an essentially dual origin for writers' cramp: a psychic element and an organic factor, the former being the more important. Patients predisposed toward this disorder are of the conventionally "emotional" or "nervous" type; only in this matrix can an organic disturbance precipitate writers' cramp. Such a person is disturbed by an injury to the right upper extremity or by a mild organic disease of the central nervous system which implicates the efficiency of that member. In his anxiety about the integrity of his handwriting he converts this ordinarily automatic procedure into a deliberate and zealously studied one.

The authors' point of view is illustrated by the citation of the cases of three patients with writers' cramp, one suffering from a scapulohumeral periarthritis, one from Recklinghausen's disease and one from osteophytic spondylitis. Each patient was of a highly emotional nature, and these three otherwise diverse people had this common factor.

Treatment of writers' cramp must therefore be directed along two lines, physical and psychic. Physiotherapy often has a happy influence on the underlying physical factor and on the peripheral physiologic disturbance. Progress in the treatment of these patients, however, depends to a great extent on the intelligence with which psychotherapy is employed.

DAVIDSON, Newark, N. J.

MODERN CONCEPTION OF THE CONVULSIVE STATES. M. OSNATO, Riv. di pat. nerv. **40**:362 (Sept.-Oct.) 1932.

Osnato reviews the modern conception of the convulsive states and emphasizes that in epilepsy many factors are involved, among which is the unknown element that produces the irritability of the brain tissue. Lowering of the threshold of excitability and sensitizing to convulsion might be the result of chemical, infectious or mechanical factors. Among chemical factors Osnato includes the toxic elements. Trauma of the brain at birth or shortly after and the infections that involve the brain in childhood initiate the establishment of epileptogenous areas. The initial process is not sufficient, however, to determine the convulsive seizure, which is brought out by alteration of the permeability of the cerebral blood vessels through a mechanism experimentally studied by Pike, Abel, Syz, Dandy and others.

When the initial factor is not an infectious or a traumatic lesion, metabolic disturbances, among which is an excessive production of lactic acid, may lead to the same result. All of the factors mentioned which lead to an increase of vascular permeability are not, however, capable as such of establishing recurrent convulsive seizures without some convulsing substance reaching the brain parenchyma. The principal characteristic of this hypothetic convulsing substance, which might be a polypeptid or any other product of the intermediary metabolism of the protein molecule with a chemical structure resembling absinthe, results in a temporary functional occlusion of the blood vessels. The repetition of such a vasospasm results in the cellular changes described by Spielmeyer and experimentally reproduced by Gildyea and Cobb. The anatomic changes are the end-results of a chain of phenomena which constitute the convulsive stage.

The elements involved in convulsive states resulting from increased intracranial pressure or from emotional or asphyxial conditions are important so far as they act mechanically or biochemically on the permeability of the blood vessels or act indirectly through influencing the autonomic innervation of the blood supply. These factors alone, however, are also insufficient to produce convulsive manifestations. For their production the intervention of a convulsive agent is absolutely essential. The nature of such an agent must be established through further biochemical investigations.

FERRARO, New York.

EPENDYMAL CYST OF THE THIRD VENTRICLE ASSOCIATED WITH DIABETES MELLITUS. F. B. BYROM and DOROTHY S. RUSSELL, Lancet **2**:279 (Aug. 6) 1932.

A woman, aged 49, had diabetes for the last twenty years of her life. Five years before death she complained of vague headaches. She had an attack of

extreme drowsiness which lasted for several days. In the last year of life she suffered from severe headache and tinnitus, which occurred a few times a day and lasted for about ten minutes. The headache began in the occipital region and radiated to the frontal region and down the spine as far as the shoulder blades. Insulin administered in increasing amounts did not control the glycosuria well. She became emotionally unstable and liable to attacks of profound depression. During the last two months of life, with a rigid diet and large doses of insulin, she remained free from glycosuria, but the blood sugar remained high. Four days before death she complained of severe headache and drowsiness; on the day of death she became comatose. Rigorous treatment for diabetic coma was without avail. The spinal fluid was under increased tension and contained an excess of protein.

At autopsy a cystic ependymal cyst of the roof of the third ventricle was found. There was markedly increased intracranial pressure due to blockage at the foramen of Monro. The pancreas showed no gross abnormality of appearance, size or weight. Microscopic examination of this organ showed no inflammatory or other lesions that would point to coincidental diabetes, independent of the cerebral condition.

Byrom and Russell stress the lack of dehydration in the comatose patient as indicative of a glycosuria that is not of pancreatic origin. They suggest that the presence or absence of gross dehydration should be regarded as evidence of the greatest diagnostic significance. It cannot be unequivocally proved in this case that the association of glycosuria and tumor of the third ventricle was not a coincidence; yet, considered in the light of the connections between the hypothalamic nuclei and the sympathetic nervous system, chronic irritation might cause a sympathetic hyperglycemia of sufficient degree and duration to initiate progressive diabetes in a patient congenitally predisposed to the disease.

BECK, Buffalo.

THE LEVINSON TEST IN TUBERCULOUS MENINGITIS. MORRIS GLEICH, Am. J. Dis. Child. **43**:1077 (May) 1932.

Gleich claims that the test, which was first reported in 1917, is of value in differentiating tuberculous from other types of meningitis. In all the cases of tuberculous meningitis that were confirmed by discovery of the bacilli and by findings at autopsy the test was positive. However, he reports some positive reactions in Levinson tests of the spinal fluids in cases of cerebrospinal syphilis, amyotrophic lateral sclerosis and uremic coma. The Levinson test is based on the principle that protein in meningitic spinal fluid reacts electronegatively or electropositively in the presence of certain alkaloid or metallic precipitants. The hydrogen ion concentration of the spinal fluid is the important factor of the test. The reaction of tuberculous and nonmeningitic spinal fluid is alkaline, while that of other types of infectious meningitic fluid is acid. The technic of the test is as follows. One cubic centimeter of spinal fluid is placed in each of two test tubes, which are 8 mm. in diameter. To one is added 1 cc. of a 1 per cent solution of mercuric chloride, and to the other, 1 cc. of a 3 per cent solution of sulphosalicylic acid. The tubes are well shaken, stoppered and allowed to stand at room temperature for forty-eight hours. At the end of twenty-four and forty-eight hours the column of precipitate in each tube is measured in millimeters. When the height of the precipitate in the first test tube is seen to be twice that of the precipitate in the second tube, the result of the test is positive.

This report is of value in that it describes the technic of one of the numerous "tests" that have come into the literature recently which may aid in differentiating tuberculous meningitis from other infections of the central nervous system. It is not an absolute differential diagnostic criterion, but it is of value when taken in conjunction with corroborative physical findings of the suspected disease.

LEAVITT, Philadelphia.

FOCAL ENLARGEMENT OF TEMPORAL BONE AS SIGN OF BRAIN TUMOR. ROSS H. THOMPSON, J. A. M. A. **99**:379 (July 30) 1932.

Thompson records and discusses two instances of external prominence of the squamous portion of the temporal bone in children, directly overlying an intracranial tumor and not due to hyperostosis but to bulging. In each, the occurrence was sufficiently early to furnish the principal evidence of the presence and localization of the growth. In one case the tumor was of meningeal origin and had no buffer between it and the thin squama directly overlying, which according to roentgen and microscopic evidence was invaded by tumor cells and thereby increasingly weakened. The dura adherent to the bone pinioned the tumor to the skull vault and created a tendency to limit direct pressure outward and inward in the immediate neighborhood. Inward pressure on the optic radiation would account for the left homonymous hemianopia. Outward pressure would account for the thinning and bulging of the bone. Pressure internally and above in the direction of the internal capsule would account for the symptoms in the pyramidal tract. The growth probably slowly decompressed itself by a gradual formation of the lump. The absence of symptoms and the improvement in the optic neuritis one year before admission probably were due to the increased cranial capacity occasioned by the bulge, although the latter was not discovered until one day before admission. In the other case the tumor did not arise from the meninges. It was of deep origin and was shown by the ventriculogram to have shifted the lateral ventricles and midline structures to the left in the direction of the opposite squama. The total lack of observable neurologic changes may thereby be accounted for by the giving way of the right squama, and also by a slighter degree of bulging of the left squama, thus creating an enlarged intracranial capacity by an increase in the cranial diameter between the two squamae.

EDITOR'S ABSTRACT.

CONTRIBUTION TO THE PATHOLOGIC ANATOMY OF RABIES IN MAN. L. ROJAS, Arch. f. Psychiat. **96**:1 (Jan.) 1932.

The author reviews the literature on this subject with special reference to the trends of interest along the lines of: (1) the type of pathologic process, (2) the localization and (3) the nature of the Negri bodies. He reports the history of the case of a man aged 25, who was bitten by a rabid dog forty-seven days before death. Prophylactic treatment was not undertaken until twenty-three days after the exposure, and the first definite signs of hydrophobia appeared seven days before death. The pathologic findings are summarized as follows: There were inflammatory as well as regressive features, the inflammatory component being characterized by a marked similarity to the findings in the acute phase of encephalitis epidemica and in the Heine-Medin disease. There were marked infiltration, both lymphatic and leukocytic, and marked neuroglial proliferation. The latter was so pronounced that it reminded one of the type of encephalitis that is encountered in measles and vaccination.

A review of other cases reported recently leads the author to conclude that polymorphonuclear leukocytic infiltration is characteristic of this disease. The localization of the process is also considered characteristic. The condition seems to show a predilection for the brain stem, the midbrain and the pallidum. In the cerebrum the inflammatory process is not so marked and is predominantly localized in the cornu ammonis and the striate body. The degenerative changes, on the other hand, are more marked in the cerebrum, especially in the cornu ammonis; in this region, too, there is a preponderance of Negri bodies, which are considered by the author to be products of degeneration.

MALAMUD, Iowa City.

OBSERVATIONS ON THE FINE NORMAL AND PATHOLOGIC STRUCTURES OF THE DENTATE NUCLEUS IN MAN. VITTORIO TRONCONI, Riv. di pat. nerv. **40**: 137 (July-Aug.) 1932.

Tronconi reviews the data concerning the embryology and histology of the normal dentate nucleus in man. He then summarizes the pathologic changes of

this structure that have been reported in various diseases of the nervous system. He describes minutely his personal observations of the dentate nucleus in twenty-five neurologic cases, including the nerve cells, neurofibrils, nerve fibers, glia, blood vessels and fat, iron and calcium content. He concludes that there are no typical lesions of the dentate nucleus corresponding with diffuse or systematized disease of the central nervous system. Probably an exception might be made for myoclonus epilepsy, in which several inclusions (Lafora bodies) have been described in the nerve elements of the dentate nucleus. In most of the cases described by Tronconi the dentate nucleus presented changes of atrophic, sclerotic or degenerative type; these changes are common in many diseases in various regions of the central nervous system. Even in cases in which clinical symptoms referable to the dentate nucleus were absent, he found more or less diffuse involvement of the nuclei. Tronconi believes that the cause of the great vulnerability of the dentate nucleus is the close relationship between the cellular elements and the blood vessels of the region. It seems that the nerve cells are closely in contact with the blood vessels and that the circulation is somewhat slowed down by the richness in veins of the dentate nucleus. Other factors, however, such as involvement of the capillaries and exogenous or endogenous elements in the sense of toxic influences, may play a subsidiary part in the determination of the particular vulnerability of the nucleus.

FERRARO, New York.

MENINGITIS AND DERMATITIS CAUSED BY A NEW VARIETY OF BLASTOMYCETE. CYRIL M. MACBRYDE and EWELL I. THOMPSON, Arch. Dermat. & Syph. **27**:49 (Jan.) 1933.

This is the first reported case of a true blastomycetic meningitis following blastomycetic dermatitis. The specific organism was the Endomyces capsulatus; only one previous case of meningitis due to this parasite has been reported, and this was not in association with a dermatitis.

The patient, a plumber, aged 28, was admitted to the hospital with several large, annular, foul-smelling ulcerations on the skin. Pus from these lesions contained the organism in a round, doubly-contoured, budding form. Two weeks after admission the patient began to complain of coryza, headache and nausea, and his temperature rose to 101 F. He then had a stiff neck and a positive Kernig sign. After another week there developed three subcutaneous abscesses, all of which yielded blastomycetes on culture. He died six weeks after the onset of the meningitic symptoms or three years after the first appearance of the cutaneous lesions. Three lumbar and three cisternal punctures had been done. Blastomycetes were found in every sample of the spinal fluid, one of the specimens from the cistern containing over 1,000 of the parasites per cubic millimeter. Globulin was present in excess in every sample, and the colloidal gold curve was 555555553. The organisms were cultured from the blood and spinal fluid, and even from the urine.

At autopsy a tenacious exudate was found on the ventral surface of the cerebellum and within the left lateral ventricle. It was made up of leukocytes, fibrin, plasma cells and giant cells, with blastomycetes in and near the latter.

DAVIDSON, Newark, N. J.

PATHOLOGY AND SYMPTOMATOLOGY OF HEADACHES DUE TO SPECIFIC SENSITIZATION. HERBERT J. RINKEL and RAY M. BALYEAT, J. A. M. A. **99**:806 (Sept. 3) 1932.

An analysis made by Rinkel and Balyeat of the symptomatology and associated pathologic changes in sixty-five patients in whom headaches could be induced by clinical trial reveals: 1. In approximately 85 per cent of the patients the headaches were characterized by prodromas, aura, unilateral or bilateral headache and a postmigrainous phase of exhaustion, sleepiness, polyuria or nasal symptoms. 2. The term migraine implies a clinical, not a pathologic entity, and should be used as such, and the specific cause of the attack should be stated, when known.

3. There is a wide variation of symptoms in the same patient, in different attacks. 4. The familial history of the headache is approximately three times as frequent as is the history of any allergic disease. 5. Headaches due to specific sensitization may be classified according to symptomatology as (*a*) migraine and (*b*) headaches without sensory, motor or vasomotor changes. The latter group includes (1) headaches without other demonstrable pathology, (2) attacks associated with allergic reactions in the nose and sinuses, that is, frontal and other headaches occurring in patients with seasonal or perennial vasomotor rhinitis, and (3) the headache of general allergic reaction. According to recurrence they may be classified as the periodic and nonperiodic. 6. The results obtained by gastric analysis, basal metabolism, cytologic study and ophthalmoscopic examination have not been of diagnostic value. The weight and height average of these patients were within the normal limits. 7. Passive transfer was accomplished, the technic of Prausnitz and Küstner being used. 8. From these studies it appears that the symptomatology, familial history and protean manifestations of headaches due to specific sensitization are similar to those of hereditary migraine.

EDITOR'S ABSTRACT.

THE ACCIDENTS OF THE MALE CLIMACTERIC. KENNETH WALKER, Brit. M. J. **1**:50 (Jan. 9) 1932.

In this paper the author emphasizes the existence of a male climacteric, or the preferable term "the critical age." In the majority of men signs of involution beginning in the organs of generation are found during the latter part of the fifth decade and the beginning of the sixth. Many of the changes are akin to those that occur in women, frequently a disposition to become fat, with its associated circulatory embarrassment. Flatulent dyspepsia is common. Hernias are common and, says the author, "It is to the male climacteric that the truss makers owe half their business." A few cases may show thinness, due to increased thyroid activity. A more or less abrupt change in appearance is common; the patient suddenly looks older.

Changes in psychologic makeup are no less pronounced than are the physical changes. Activity is inhibited, and conservatism becomes evident. Frequently, a certain amount of emotional instability is seen, with a tendency to a sudden flare-up in the dying embers of desire, sometimes associated with what is termed the "gerocomic tendency," that is, the desire of an elderly man for very young girls. Exhibitionism may be an associated manifestation of this complex. Changes in the reproductive organs themselves are pronounced, usually prostatic involution, though occasionally the reverse, hydrocele, fibrosis of the testes, multiple retention cysts in the epididymis and frequently changes in the seminal vesicles.

Treatment of the male climacteric is for the most part symptomatic and dependent on the particular pathologic change present. The author discourages any attempts at endocrine therapy.

FERGUSON, Niagara Falls, N. Y.

SUPERIOR PULMONARY SULCUS TUMOR: TUMOR CHARACTERIZED BY PAIN, HORNER'S SYNDROME, DESTRUCTION OF BONE AND ATROPHY OF HAND MUSCLES. HENRY K. PANCOAST, J. A. M. A. **99**:1391 (Oct. 22) 1932.

Pancoast reports seven cases of a peculiar neoplastic entity found in the upper portion of the pulmonary sulcus of the thorax. He previously reported three cases and adds four more recent ones. The tumor is evidently epithelial in its histopathology, but its exact origin is uncertain. It seems likely that it may arise from some embryonal rest. Its point of origin is always the same, and it produces constant and characteristic clinical phenomena of pain in the eighth cervical and first and second thoracic trunk distribution, wasting of the muscles of the hand and Horner's syndrome. The roentgen observations are typical, consisting of a small, sharply defined shadow in the apex of the thorax, destruction of one or all of the upper three ribs in their posterior aspects and the adjacent transverse processes, and sometimes slight erosion of the vertebral body. So far this tumor has resisted all efforts at irradiation treatment. It is obviously not subject

to surgical removal, although it is accessible. It is rather rapidly fatal. In six cases death ensued in from eleven to fourteen months after the onset of symptoms. In one more or less doubtful case it occurred in four months. The cause of death was uncertain. There must have been metastases somewhere, but none was found in the lungs or mediastinum. In one case the liver was enlarged. Unfortunately there were no autopsies. The author believes that in the light of present knowledge, the only possible benefit would be irradiation carried out as in any other spinocellular carcinoma. This means ten or more erythema doses delivered by external irradiation plus radon implants after surgical exposure.

EDITOR'S ABSTRACT.

WHAT THE OPHTHALMOLOGIST SHOULD EXPECT OF THE NEUROSURGEON IN COMPRESSION OF THE CHIASM AND OPTIC NERVES. C. VINCENT, Ann. d'ocul. **169**:558 (July) 1932.

In his communication Vincent considers especially cases in which there may be compression of the chiasm and optic nerves. He presents five cases in which recovery occurred; in one case the patient had been operated on three times and in another two times. The visual acuity and fields of vision were greatly improved in these cases. The cause of the compression was a chromophilic adenoma of the hypophysis, a tumor derived from Rathke's pouch, a cystic tumor, a suprasellar meningioma and a case of retrochiasmatic arachnoiditis. With the presentation of these cases, Vincent reported his statistics on operations from Dec. 1, 1929, to May 1, 1932, bearing on 72 retrochiasmatic interventions. The average mortality is 11.1 per cent (8 deaths). Of the 72 cases in which operation was performed, there were 27 adenomas of the hypophysis (1 death), 8 tumors of Rathke's pouch (2 deaths), 5 meningiomas (1 death), 17 cases of retrochiasmatic arachnoiditis (3 deaths), 4 gliomas of the chiasm (1 death), and 1 case each of retinocystoma, meningioma and abscess. Seven explorations of the third ventricle were performed without mortality. Certain of these patients were completely cured or improved, but in others there was no improvement of visual acuity, because the optic nerves were already atrophied at the time of intervention. Vincent calls attention to the fact that the true state of the optic nerve could not always be determined from the ophthalmoscopic appearance. He believes in the necessity for early operation, and that the operative risks are not very great.

BERENS, New York.

RESPONSE OF TACTILE RECEPTORS TO INTERMITTENT STIMULATION. MCKEEN CATTELL and H. HOAGLAND, J. Physiol. **72**:392, 1931.

The authors utilized a preparation of the dorsal cutaneous nerves of the frog which enabled them to record the action potentials in single nerve fibers from receptors in the skin. Using an interrupted air blast as stimulation, it was possible to study the response to a rapid sequence of stimuli. It was found that the receptor produces a single impulse in response to each brief puff of air up to a high frequency of stimulation. The adaptation time to continued intermittent stimulation varies with its character: If the duration of the stimulus (the puff of air) is long in relation to the intervening periods of rest, adaptation occurs within a few seconds or minutes, whereas when the relative durations of these factors are reversed the receptors may continue to follow a high stimulation rate for an hour or more.

When a continuous air blast is applied to the receptors, even when no response is elicited, it results in complete adaptation to an immediately following intermittent stimulus; i. e., adaptation may occur as the result of the stimulus without the production of a single impulse. Impulses reaching the end-organ antidromically cause a decrease in its excitability to subsequent stimulation, resembling that occurring when the stimulus is applied directly. The receptors may respond to closely spaced stimuli at a rate bringing successive impulses in the nerve fiber well within the refractory period. The action potential of the second impulse recorded from the single nerve fiber is then reduced in magnitude.

ALPERS, Philadelphia.

NEXT STEPS IN A MENTAL HEALTH CAMPAIGN. SANGER BROWN, Psychiatric Quart. **6**:411 (July) 1932.

Having completed its first step — the transfer of patients from the brutality of the old-fashioned insane asylum to the scientific care of the psychopathic hospital — psychiatry is now ready to enter its second phase: the prevention of mental disease. Much of the pessimism on the subject is unnecessary and is comparable to the attitude taken toward tuberculosis several decades ago. In this instance, although no specific contribution to therapy has been made, the reath rate has been lowered from 194 to 67 in the last thirty years. The keystone in mental hygiene is the education of the public. It is the ten or fifteen years before a mental breakdown becomes obvious that is of greatest importance in psychologic prophylaxis, and the public must be taught to come to the psychiatrist during that period. There is no reason why mental hygiene should not be taught in schools as carefully as French verbs or as practically as the care of the teeth. The adult public should be educated through bulletins, radio talks, lectures and newspaper articles. The psychiatrist will be able to show the worried patient that the repressions and conflicts which precipitate his suffering are often unnecessary; he will be able to explain that other persons have been relieved of similar problems; he will be ready to present proper methods of thinking and behaving, just as the public health physician is able to teach proper posture and proper diet; the results should be equally happy.

DAVIDSON, Newark, N. J.

A SEARCH FOR TROPHIC INFLUENCE OF POSTERIOR SPINAL ROOTS ON SKELETAL MUSCLE, WITH A NOTE ON THE NERVE FIBRES FOUND IN THE PROXIMAL STUMPS OF THE ROOTS AFTER EXCISION OF THE ROOT GANGLIA. SARAH S. TOWER, Brain **54**:99 (April) 1931.

The posterior root ganglion of the muscles of one forelimb was removed in four cats, and in one of these the stellate ganglion also. The animals were kept alive for from four months to one year and examined as to sensation, motor activity and trophic status. In the cats with dorsal root ganglionectomy, the weight of gross fixed specimens, the microscopic appearance of the muscle fibers and the measurements of 600 fibers were essentially not different from those of normal specimens. Sympathetic ganglionectomy, in addition to section of the dorsal root, was no more effective than the first procedure in producing atrophy or dystrophy of skeletal muscle. A study of the nerve fibers present in the proximal roots after excision of the root ganglia confirmed the opinion of Sherrington and others that these are regenerating fibers. The data do not support the theory of Kuré that the posterior roots and ganglia, with or without assistance from the sympathetic nervous system, are concerned in maintaining the normal trophic state of skeletal muscle, and that their destruction leads to atrophy and dystrophy. The author raises the question of the necessity of the concept of "trophic state." It can be concluded that the trophic state of skeletal muscle seems to be dependent solely on motor innervation.

MICHAELS, Boston.

TREATMENT OF STUPOR. KARL LANGENSTRASS, Am. J. Psychiat. **11**:447 (Nov.) 1931.

Combining the carbon dioxide oxygen inhalation technic of Lövenhart with artificial fever therapy, Langenstrass obtained good results in the treatment of patients with catatonic stupor. The series consisted of twelve physically healthy young adults in all of whose cases a diagnosis of catatonic dementia praecox had been made. (In the discussion Karl Bowman challenged the diagnosis in three of the cases.) During the three weeks preceding the treatment by inhalation, the patients received intravenous injections of diluted erysipelas and prodigiosus toxins (Coley); the dose was ⅛ minim (0.007 cc.) on the first day and was increased gradually to 3 minims (0.18 cc.) on the twenty-first day. Injections were given at two day intervals. After the course of fever was completed, each patient was subjected to inhalations of gas, a mixture of carbon dioxide and

oxygen being used. The percentage of carbon dioxide was slowly increased from 0 to 20 per cent in ten minutes and was sustained at that level for a quarter of an hour. Six of the twelve patients showed a period of responsiveness of less than four hours; two were free from stupor for a week, and four were responsive for many months. Langenstrass believes that this combination of inhalation treatment with fever therapy represents a useful intensification of the Lorenz-Lövenhart procedure.

DAVIDSON, Newark, N. J.

THE EFFECTS OF OPTIC STIMULI UPON THE FORMATION AND DESTRUCTION OF MELANIN PIGMENT IN FISHES. F. B. SUMNER and N. A. WELLS, J. Exper. Zool. **64**:377 (Feb. 5) 1933.

When specimens of the guppy, Lebistes reticulatus, are transferred from a light to a dark background, new melanophores appear to be formed in situ, apparently from cells belonging to the dermis overlying or underlying the scales. They are never formed in the epidermis. In specimens that have been transferred from black to white, degenerating melanophores are extruded in great numbers through the epidermis to the exterior. These processes take place much more slowly than the familiar prompt changes of shade, which are due to the rearrangement of the pigment present without change in its amount. They are manifest to some extent within a few days and are completed within a few weeks. Apparently optic stimuli may have some definite trophic effects on the chromatophores, leading to an increase in the production of melanin pigment or to its elimination. Production of guanine appears to be stimulated by the same optical conditions that lead to the suppression or elimination of melanin.

Even the most prolonged exposure to a given background fails to render the resulting pigmental condition permanent. Fishes reared from birth in black or white jars respond fairly promptly to a reversal of backgrounds.

WYMAN, Boston.

THE OCCURRENCE OF ATTACK-LIKE COMPULSIVE PHENOMENA IN THE COURSE OF POSTENCEPHALITIC PARKINSONISM. W. CHLOPICKI, Arch. f. Psychiat. **93**:1, 1931.

The author reports six cases of postencephalitic parkinsonism in which there occurred attacks of ocular spasms accompanied by compulsive phenomena. In two cases, the compulsions occurred before the ocular spasms; in the other four, they coincided with them. The author discusses the relationship of the psychic phenomena to the eye movements and the possible organic basis of both. Fundamental to the compulsive phenomena, according to Chlopicki, is the primary organic anxiety state. The phenomenon of the blocking of the processes is regarded as analogous to iterations of speech and of other acts. The ocular spasm is probably due to a blocking in certain extrapyramidal tracts, which brings about a release from the braking influence of higher centers. The exact reasons for the occurrence of these compulsive phenomena in certain persons, and their time relationship to the spasms of the eyes must be looked for in the personality of the patient.

The author is of the opinion that the patients are constitutionally of a type susceptible to compulsive behavior. Although one cannot differentiate definitely these compulsive phenomena from those that occur on the basis of functional disturbances, they show some differences in that they are simpler and less systematic.

MALAMUD, Iowa City.

CEREBELLUM OF TRITURUS TOROSUS. O. LARSELL, J. Comp. Neurol. **53**:1 (Aug.) 1931.

The cerebellum of Urodeles represents a stage of development that lies between the primitive condition of the generalized fishes and the cyclostomes, on the one hand, and of the more complex and modified condition of anurans and reptiles

on the other. The cerebellum of Triturus is composed of a central corpus cerebelli, derived from the rostromedial part of the rhomboidal lip, and of bilateral auricular lobes which are intimately associated with the acusticolateral areas of the bulb and with the body of the cerebellum. Histologically, it shows the transition from the relatively simple structure of the medulla oblongata to the differentiated pattern of the cerebellum. The small cells of the corpus cerebelli are definitely granules. Its molecular layer is continuous around the rostroventral and lateral part of the auricle, with the layer of fibers and neuropil of the acusticolateral area. This layer of the acusticolateral area corresponds to the crista cerebellaris of fishes. There are two sets of commissural systems: (1) the cerebellar commissure, which includes intertrigeminal fibers, direct trigeminal fibers and spinocerebellar fibers, and (2) the vestibulolateral commissure, which appears to be the path along which cell elements of the auricular lobes migrate medially to form the basis of the posterior lobe of the cerebellum of higher forms.

ADDISON, Philadelphia.

EXOPHTHALMOS OF CENTRAL ORIGIN. W. RUSSELL BRAIN, Brit. M. J. **2**:937 (Nov. 21) 1931.

Ten cases are reported from the author's series of between twenty and thirty cases illustrative of exophthalmos of central origin. The lesions indicate a midbrain location. None of the cases represents postmortem material. The author enumerates the following clinical features, somewhat in the manner of a classification. 1. Exophthalmos may be associated with: (*a*) supranuclear ocular palsies, especially paralysis of conjugate vertical movement and convergence; (*b*) external ophthalmoplegia—some of these cases are etiologically obscure and exhibit bizarre defects of ocular movement and (*c*) internal ophthalmoplegia—in one case unilateral exophthalmos was associated with an Argyll Robertson pupil in the same eye; in another the reaction to light was sluggish in both eyes. 2. Exophthalmos may be unilateral or bilateral, and if bilateral it may be equal or unequal in degree on the two sides. 3. It may be associated with retraction of the lid or with ptosis, either of which, again, may be unilateral or bilateral, and equal or unequal. There may be retraction of the lid on one side and ptosis on the other. 4. Exophthalmos may occur with or without enlargement of the thyroid. 5. It may occur with or without disturbance of sugar metabolism.

FERGUSON, Niagara Falls.

NEUROFIBRILLAR STRUCTURE OF THE SPINAL CORD OF CAT EMBRYOS CORRELATED WITH THE APPEARANCE OF EARLY SOMATIC MOVEMENTS. WILLIAM F. WINDLE, J. Comp. Neurol. **53**:71 (Aug.) 1931.

This article is an attempt to correlate the development of fetal behavior with microscopic pictures. In a previous communication, observations on the development of motility in cat embryos and fetuses were reported. In this paper the author considers the neurofibrillar structure of the spinal cord in these same embryos. Most of the embryos were prepared by the pyridine-silver technic of Ranson; some by the Bielschowsky method. In the premotile specimens of from 10 to 14 mm., cell elements necessary for primitive reflex pathways were present, but almost none of the sensory neurons were associated by means of collaterals with the mantle layer neuroblasts. The earliest motion appeared in embryos 16 mm. long. At this time well formed sensory collaterals are seen, and there is a direct relationship between their number and size and the regions in which movements were seen. The first sensory collaterals appear at the ventrolateral end of the posterior funiculus, and not until movements are well established are they found in any numbers toward the dorsomedial end. Further growth follows the cephalocaudal plan, as does the development of motility.

ADDISON, Philadelphia.

A Study of the Relationships Between Compulsion Neurosis and Paranoia. E. Stengel, Arch. f. Psychiat. **95**:8 (Aug.) 1931.

The author reports two cases, one of a woman aged 24 and the other of a man aged 35, to whom she was engaged. The woman came to the clinic with complaints of compulsive thoughts and acts which she had had for three years. These were centered on the idea that she was sexually promiscuous with different men, and that she had practiced perversions with them. During the process of the development of these thoughts she began to have a handwashing obsession. The man, who had always been of a suspicious nature, developed, on the basis of his fiancée's compulsive thoughts, the paranoid idea that she actually did practice perversions with a number of men. A vicious circle was thus established, the compulsive phenomena being further increased by the accusations of the man, whose paranoid ideas kept gaining strength on the basis of the woman's compulsive thoughts and acts. The early experiences and constitutional anomalies of these two patients and the effect they had in bringing about the development of the diseases are discussed. The literature on the subject and concerning the general concept of induced psychoses is also discussed.

Malamud, Iowa City.

Innervation of the Pia Mater of the Spinal Cord and Medulla. Sam L. Clark, J. Comp. Neurol. **53**:129 (Aug.) 1931.

Nerve fibers and endings in the spinal and medullary pia mater of nine adult cats were stained with pyridine silver. After staining and while still in 95 per cent alcohol, the pia was stripped from the cord, mounted on microscopic slides, dehydrated and mounted again. As many as thirty nerve bundles were reported in the cervical region. The majority of these ran parallel to the long axis of the cord. In general, the course of the bundles is independent of the vascular channels. These bundles contain many unmyelinated and a few myelinated fibers. The origin of all the nerve fibers in the spinal pia mater is not known. No ganglion cells have been observed in the spinal pia, though a few appear in the pia over the cerebral peduncles. Exclusive of vasomotor nerves to the muscle of the arterial system, the nerve fibers end on the pia itself and on or near blood vessels in terminations of "sensory" type. Nerve fibers apparently enter the spinal pia directly from the white matter of the cord, as well as from both dorsal and ventral roots of the spinal nerves. The arrangement of nerve fibers in the pia mater of the medulla oblongata is similar to that of the spinal cord.

Addison, Philadelphia.

Mental Hygiene and the College. Frankwood E. Williams, Ment. Hyg. **15**:532 (July) 1931.

Mental hygiene has always been practiced in the college. The sage and sympathetic professor who advised worried students was practicing mental hygiene years before the phrase was ever created. But mental hygiene as practiced by a psychiatrist is a sharper, more effective technic. The trained psychiatrist differs from the sagacious adviser drawn from academic circles in several significant particulars. His method is more objective; he attaches less significance to intellectual attainments; he hears between the lines spoken to him and does not accept statements at their face value. He knows that the complaint of the worried student is often but the rationalized coating of a greater and quite different problem. When he sees sudden cures follow sympathetic interviews he does not assume with smug complacency that the problem has been solved; he is as suspicious of these results as of his students' complaints in the first instance. He has keener prognostic skill, and can differentiate more accurately between benign and malignant situations. Williams closes with a plea for better clinical training in college mental hygiene.

Davidson, Newark, N. J.

IMMUNITY TO HUMAN AND PASSAGE POLIOMYELITIS VIRUS. SIMON FLEXNER, J. A. M. A. **99**:1244 (Oct. 8) 1932.

Flexner reports that the passage of the virus of poliomyelitis through monkeys intensifies its infectivity for these animals and modifies its specific immunologic reactions. The changes undergone are quantitative and probably also qualitative in nature. The modified virus preserves its immunizing power, in part at least, against recent strains of the human virus. Immune serums prepared with human and with monkey passage strains of virus exhibit quantitative differences in cross-neutralizing tests. Virus-serum immunity can be produced in monkeys by the separate, simultaneous injection of one or more doses of virus and immune serum. The dangers of active poliomyelitic symptoms arising in the inoculated animals seem lessened by the combined treatment. The optimum manner of producing virus-serum immunity has still to be determined. Monkeys that were proved to be wholly refractory to nasal instillations of the virus were shown by the author to be devoid of serum antiviral activity and to exhibit average susceptibility to intracerebral inoculation of potent virus.

EDITOR'S ABSTRACT.

DATA ON THE RELATIVE TIME OF FORMATION OF THE CEREBRAL GANGLIA OF AMBLYSTOMA JEFFERSONIANUM. F. L. LANDACRE, J. Comp. Neurol. **53**:205 (Aug.) 1931.

For this study a series of Amblystoma containing fifty-seven stages from three clutches was used. The ganglia studied were neural crest, dorsolateral and epibranchial. The ganglia form between 222 and 316 hours after deposition. The relative time of formation of cranial ganglia in Amblystoma is based on criteria which are different for neural crest ganglia as compared with lateralis and epibranchial ganglia. The criteria for the earliest stages of dorsolateral and epibranchial ganglia include the earliest stage at which placodes undergo a definite proliferation, and the latest stage is placed at the time when the placode becomes detached from the ectoderm. The earliest stage of the neural crest ganglion is placed at the time when the condensed ganglion can be distinguished from the loose mesenchyme derived from the neural crest. The last stage is placed at the time when the ganglion cells elongate, indicating probably the formation of cell processes.

ADDISON, Philadelphia.

RESOCIALIZING TENDENCIES IN PHYSICAL EDUCATION FOR THE MENTALLY ILL. J. E. DAVIS, Ment. Hyg. **15**:354 (July) 1931.

The activities of play constitute, Davis believes, a stimulus to social consciousness of unique effectiveness. An athletic game dissipates in action the feelings of "social sickness"; a desire to win may be goal enough to keep a patient interested in group activities. Games engender in a concrete way such socially useful feelings as willingness to cooperate with others, sense of team loyalty, willingness to subordinate self to the group, respect for the property of others, self-realization (through applause), appreciation of fair play and the necessity for delegating authority. By way of illustration, he cites the case of a baseball player who was shamed, more or less reluctantly, into playing for his ward team. Davis repeatedly emphasizes, however, the need of rapport between the physical educator and the patient; an attitude of self-confidence between pupil and teacher will dissipate in some measure the unfavorable traits of timidity, fear and distrust.

DAVIDSON, Newark, N. J.

BATH TREATMENT FOR DEAFNESS. JAMES ADAM, Brit. M. J. **1**:621 (April 11) 1931.

Six cases of chronic progressive deafness are reported, along with two cases of Ménière's disease. Treatment consists of hot baths containing a pound of commercial magnesium sulphate. One infers that the frequency of the baths is every other day the first week, once during the following week, then once every

fortnight. Ten or fifteen minutes' duration is advised. Startling results are reported in some of the cases. One patient suffering from Ménière's complex was unimproved; the other showed pronounced improvement but only after the quantity of magnesium sulphate was doubled.

The author asserts that this treatment is as yet in the tentative stage, worthy of trial, but likely to succeed only in a minority of chronic cases. It fails when it fails to sweat the patient. It is not certain that magnesium sulphate is essential.

FERGUSON, Niagara Falls, N. Y.

FURTHER OBSERVATIONS ON THE COMPONENTS OF THE MYENTERIC PLEXUS. SYDNEY E. JOHNSON and MARTIN PALMER, J. Comp. Neurol. **53**:169 (Aug.) 1931.

The purpose of this paper was to determine whether the small interstitial cells found in the myenteric plexus were glia cells, as Trostanetzky called them, or cell bodies which give rise to a fine, primitive network, as Lawrentjew maintains, or whether they are ordinary connective tissue cells, as one of the authors reported them previously. Material from frogs, cats and dogs was prepared by a variety of connective tissue, neuroglial and silver methods. In the three forms studied the only elements found were the large neurons, vascular tissue, smooth muscle and connective tissue. The so-called interstitial elements were found to stain by the differential connective tissue methods. The authors found no evidence of glia cells or of a primitive nerve mechanism.

ADDISON, Philadelphia.

A STUDY OF THE ETIOLOGY OF MULTIPLE SCLEROSIS. ARTHUR WEIL, J. A. M. A. **97**:1587 (Nov. 28) 1931.

Weil describes experiments in which the attempt was made to demonstrate the presence of a filtrable virus in cultures from spinal fluids of patients with multiple sclerosis, as reported by Chevassut and Purves-Stewart. The repetition of the experiments of these investigators failed to produce convincing evidence that, in multiple sclerosis, cultures from spinal fluids yield a filtrable virus and that this virus is responsible for the production of the disease. The author believes that the fact that spheres and colonies of spheres may more readily be seen in agar cultures of spinal fluids that have given a positive globulin reaction suggests the precipitation of colloidal protein (or lipid) particles, which become visible in the dark field.

EDITOR'S ABSTRACT.

PSYCHIATRIC REACTIONS FROM DYSINSULINISM IN A DIABETIC PATIENT. HERBERT J. CRONIN, J. Nerv. & Ment. Dis. **74**:478 (Oct.) 1931.

The case of a college girl, aged 21, with definite evidence of constitutional inferiority, feelings of inadequacy, inability to face reality, fears of the future and a desire to remain in a state of protection and support is described in detail. Prior to the discovery of glycosuria and high blood sugar, she had given a history of diplopia of two or three weeks' duration. After diabetic treatment with 15 units of insulin daily, diplopia recurred with mild confusional states. Analysis with the object of uncovering some hidden psychic material of traumatic character was given up as impossible. Subsequent attacks of diplopia were followed by a stuporous state in which silly, childish, assaultive activity, with delusions of poisoning and negativism suggesting an early schizophrenia, developed. The exact relationship of the mental phenomenon to the use of insulin is, however, not made clear.

HART, Greenwich, Conn.

AMAUROTIC FAMILY IDIOCY. LUDO VAN BOGAERT, J. SWEERTS and L. BAUWENS, Encéphale **27**:196 (March) 1932.

In presenting two cases of amaurotic idiocy, the authors comment on the paucity of studies of this sort in the French literature. The immediate genealogy of the

two cases is well worked out. One patient, a girl, who died at 13 months, was the child of a woman whose brother was father to the other patient. One of the cases lacked the typical ophthalmologic modifications but presented the mental picture of idiocy. In both cases the syndrome of decerebration was completely developed (tonic attacks, tonic neck and labyrinthine reflexes, decerebrate attitudes, etc.). Between the tonic contractions there existed a general state of hypotonus, with intense reflex response to touch and hearing. Palmar and plantar reflexes were inverted, i. e., of a fetal stage of development. Also in one case certain rhythmic automatic movements suggested fetal movements. ANDERSON, Los Angeles.

SENSORY COMPONENTS OF THE SPINAL ACCESSORY NERVE. WILLIAM F. WINDLE, J. Comp. Neurol. **53**:115 (Aug.) 1931.

Cat fetuses prepared by pyridine silver or the Bielschowsky technic were used to follow the course of the accessory nerve. Serial sections of the eleventh pair of cranial nerves were made in two monkeys and two cats, after fixing in van Gehuchten's fluid. Toluidine blue was the stain used. Clusters of cells and scattered single cells were found in all animals extracranially, as well as intracranially. After sectioning the spinal accessory on one side, the cats were allowed to live for from eight to twelve days. The first four cervical ganglia and the accessory nerves were examined histologically for chromatolysis. A large number of cells of the first cervical ganglion, as well as the cells on the eleventh nerve, showed chromatolytic changes. It is most probable that this sensory component is proprioceptive in function. ADDISON, Philadelphia.

THE PHILOSOPHY AND OBJECTIVE AIMS OF MODERN EDUCATION. FREDERICK L. PATRY, Ment. Hyg. **15**:550 (July) 1931.

The traditional aims of education have always been to provide a grasp of certain more or less academic subjects and to bestow the stamp of culture. Modern education, however, does more than this. Not only are the talents of the child considered, but his handicaps, too, are taken into account; an effort is made not merely to prepare a theoretically average child for a theoretically average life, but rather to enable each pupil to adjust himself to life as he finds it. The best criterion of the correctness of this new attitude is to be seen in its concrete results. Stress should not be laid on process alone, nor should emphasis be concentrated on methodology. In the last analysis, it is by the adequateness of their performance that the newer methods must stand or fall.

DAVIDSON, Newark, N. J.

EXPERIENCES WITH ENDOLUMBAR INTRODUCTION OF AIR IN TRAUMATIC HEADACHE. G. H. MONRAD-KROHN, Acta psychiat. et neurol. **6**:395, 1931.

The author has tried Penfield's method of endolumbar injection of air in nine cases of traumatic headache. In two there was complete recovery; in two, great improvement; in one, doubtful improvement; in three, no change, and in one the condition seemed to be worse following treatment. Monrad-Krohn finds the procedure painful to the patient and often accompanied by vomiting. Relief comes after from two to four days. The author thinks that his results are not so good as Penfield's, perhaps because eight of the nine patients were insured and had a pecuniary interest in the headache. He believes, however, that the method should be tried in headaches of long standing following injuries to the head.

PEARSON, Philadelphia.

CONGENITAL OCULAR TORTICOLLIS. J. LÓPEZ LACARRERE, Ann. d'ocul. **168**:563 (July) 1931.

Congenital torticollis of ocular origin has been studied by Lacarrere. He calls attention to the fact that tilting of the head is usually due to vertical deviation

of the eyes. In a patient seen by him there was no vertical deviation, but there was marked tilting of the head due to a spasmodic esotropia which was compensated for by tilting the head. The child's head was tilted at an angle of 30 degrees, which he points out as usually associated with vertical strabismus of 20 degrees. In conclusion he calls attention to the fact that the muscles of the neck have been operated on for torticollis, which was really compensatory for a deviation of the ocular muscles, and the operation should have been performed on the muscles of the eye.

BERENS, New York.

THE DANGERS AND ADVANTAGES OF SEX INSTRUCTION FOR CHILDREN. KARL DE SCHWEINITZ, Ment. Hyg. **15**:561 (July) 1931.

Facts represent no prophylactic against sexual misbehavior, and merely telling the child the details of sexual function is no assurance that he will have no maladjustments in this field. De Schweinitz pleads for a less somber, less pedantic, less serious approach to the problem. There is, he insists, no need of holding an interview when the "facts of life" are told to an embarrassed child by a still more embarrassed parent. Rather, let the child's questions be answered naturally and casually when they arise. He expresses the formula for happy child guidance as being an instruction to parents to "love them, set them a good example, and leave them alone."

DAVIDSON, Newark, N. J.

IMPRESSIONS AND OBSERVATIONS RECEIVED DURING THIS YEAR'S EPIDEMIC OF POLIOMYELITIS IN FINLAND. ELIS LOVEGREN, Acta psychiat. et neurol. **6**:327, 1931.

The author presents a brief study of the general course of the epidemic of poliomyelitis in Finland. His most interesting observation is that in one area the disease seemed to be spread largely by contact, but in another district its spread seemed to follow the course of the rivers. A similar spread was not observed in connection with railroads or other means of communication unless they lay along streams. He raises the question as to whether some cases of poliomyelitis are not water-borne.

PEARSON, Philadelphia.

ALTERATION OF DECEREBRATE RIGIDITY BY SECTION OF NERVE SUPPLY. M. NAKANISHI and T. HAYASHEDA, Keijo J. Med. **2**:483 (Dec.) 1931.

The authors describe in decerebrated cats a residual contraction of the muscles after the nerves of supply have been sectioned. They conclude that the inhibition of decerebrate rigidity stands in no relation to the sympathetic nerves. Ipsolateral sensory nerve stimulation of the muscle of the posterior extremities produces a diminution of the decerebrate rigidity following a fibrillary type of muscular contraction which is termed a release phenomenon (*Loschphänomene*).

HART, Greenwich, Conn.

RESULTS OF MEASURING BINOCULAR VISION IN REDUCED ILLUMINATION ON SUBJECTS WITH NORMAL RETINAS. E. HAAS, Ann. d'ocul. **169**:146 (Feb.) 1932.

With the aid of his "photoptometer," Haas measured the minimum illumination necessary to permit visual acuity of 1/10. A series of forty measurements shows that the results are remarkably constant in normal subjects. Consequently, in all cases in which the visual acuity in reduced illumination is subnormal, a definite functional or anatomic anomaly of the retina should be suspected.

CLINICAL, PATHOLOGIC AND THERAPEUTIC CONSIDERATIONS OF SCHIFF'S OBSERVATIONS ON "THE SPONTANEOUS EVACUATION THROUGH THE NASAL PASSAGES OF A CYST OF RATHKE'S POUCH." M. BOURGUET, Ann. d'ocul. **168**:933 (Nov.) 1931.

Bourguet reports the cure of a cyst of Rathke's pouch through the spontaneous evacuation of the contents of the cyst through the nose. In attacking this type of cyst, Bourguet says that he prefers the endonasal route. Hartmann, in discussion, called attention to the fact that the present tendency of the most prominent

The measure of physiologic reserve or cardiovascular efficiency represented by the endonasal route.

CYSTIC CAPILLARY ANGIOMATOSIS OF THE RETINA. J. MAWAS, Ann. d'ocul. **168**:890 (Nov.) 1931.

A complete study of angiomatosis retinae has been made by Mawas. He calls attention to the association of this disease with Lindau's disease and also states that there are certain analogies between Coats' disease and angiomatosis retinae. The paper is accompanied by a complete bibliography of the literature from 1879 to 1930.

A CASE OF UNILATERAL EXOPHTHALMOS WITHOUT SENSORY DISORDERS, ASSOCIATED WITH CAROTID-JUGULAR ANEURYSM OF THE SAME SIDE. WORMS, Ann. d'ocul. **169**:145 (Feb.) 1932.

Worms reports a case of unilateral exophthalmos due to carotid-jugular aneurysm of the same side without sensory disorders.

BERENS, New York.

Society Transactions

NEW YORK NEUROLOGICAL SOCIETY AND NEW YORK ACADEMY OF MEDICINE, SECTION OF NEUROLOGY AND PSYCHIATRY

Joint Meeting, Jan. 10, 1933

BRYON STOOKEY, M.D., *Chairman of the Section, Presiding*

CIRCULATORY DISORDERS IN PSYCHONEUROSIS IN TERMS OF THE SCHNEIDER INDEX. DR. JAMES H. HUDDLESON, DR. S. E. SOLTZ (by invitation) and DR. PAUL HAUN (by invitation).

Two hundred case histories of patients studied in the Neurological Institute of New York by different physicians were surveyed for a comparison of diastolic blood pressure and pulse pressure. A psychoneurosis had been diagnosed in all cases, and other conditions that might disturb the circulatory balance had been excluded. Under ordinary conditions, it was determined that the diastolic blood pressure and the pulse pressure of psychoneurotic patients as a group did not seem to vary significantly from those of normal subjects. The elimination of variables under "basal" conditions may offer new possibilities.

The measure of physiologic reserve or cardiovascular efficiency represented by the Schneider index, while far from being specific for psychoneurosis, offers a rough estimate of physical disability associated with the neurosis, provided that other pathologic influences have been considered or excluded. The mean Schneider index of a preliminary group of psychoneurotic patients examined by us was found to be plus 8.5, a score 1.5 below the accepted minimal normal score of plus 10. In 60 per cent of the cases examined the score was below plus 10.

DISCUSSION

DR. LOUIS CASAMAJOR: I have had no experience with the Schneider index, and my point of view is altogether out of sympathy with what Dr. Huddleson has presented. He has assumed that a psychoneurosis is something. I do not believe that a psychoneurosis is anything. It is not a clinical concept; one cannot compare a psychoneurosis with such a condition as pernicious anemia or carcinoma of the stomach. I think that what we need is a new term to describe, not the condition of psychoneurosis, but rather the psychoneurotic way of thinking. It is a type of mind, a type of reaction, and as the result of the particular type of reaction, which may be more or less forced on the subject by his psychologic development, it appears as a group of symptoms. One finds the psychoneurotic symptoms in the psychic spheres, as in the anxieties. Second, there are psychoneurotic symptoms in the motor sphere, as in the conversion neuroses. Third, there are the psychoneuroses which express themselves in the sensory sphere in anesthesias and dysesthesias, and last, there is the group of psychoneuroses manifesting their activity in the sphere of the autonomic nervous system. I assume that Dr. Huddleson is talking about the psychoneuroses which find their expression in the sympathetic nervous system. It is important, I think, to differentiate these cases from the point of view of prognosis. In his experiments Dr. Huddleson has apparently been trying to find some index of physical disability. That, of course, is of value, and if one could have such an index of disability, one would have something well worth striving for. As a general rule, the disability in the cases having their expression in the field of the sympathetic nervous system is

greater than in any other. Less severe are the sensory cases, even less so are the motor cases, and often, so far as physical disability goes, those that have their expression in the psychic field are the least severe of all. If Dr. Huddleson is trying to present a means of approach to the cases that have their expression in the sympathetic field (which include not only the cardiac and vasomotor cases but also the gastric cases and the instances of mucous colitis) he might provide something of value. I should like to see such a study carried on with more definitely selected cases rather than on psychoneurotic patients as a group. The patients should be selected from the point of view of sympatomatic expression; then if one found that the cases with definite autonomic and sympathetic symptoms showed a difference in the Schneider index, perhaps this would be a valuable means of approach to the problem not of the psychoneuroses but of the accompanying physical disability.

Dr. George H. Hyslop: In Dr. Huddleson's presentation a side-light was thrown on a point in terminology which I should like to see receive further attention. The so-called effort syndrome is in some respects similar to the condition generally known as neurasthenia. For the past twenty years there has been a tendency to regard neurasthenia as of psychoneurotic or psychogenic origin. It has been my opinion that neurasthenia is a term applied to a clinical syndrome; it should not be referred to a single cause. In the evaluation of the Schneider index, I think that Dr. Huddleson has reminded us that somatic symptoms and the associated psychic disorders in neurasthenia are often of common origin, regardless of whether the etiologic factor is toxic, constitutional or psychogenic. The use of the word neurasthenia offers frequent grounds for argument in questions of terminology and diagnosis; it is commonly used in the field of industrial disease and disability. It is time that we have a clarified opinion as to what neurasthenia means.

Dr. Joshua Rosett: I wish to ask Dr. Huddleson whether the reverse is likewise true, that is, whether patients with cardiovascular defects are afflicted with the symptoms of psychoneurosis.

Dr. Philip John Trentzsch: This paper is of interest in that it makes an attempt to correlate the organic with the psychogenic. In testing a group of psychoneurotic patients I do not know how one could easily make a selection. It would be much better to attack the problem and to make the selection later. Experiments with the Schneider index in the psychoses showed in 75 per cent of catatonic and hebephrenic patients a low rate similar to that reported by Dr. Huddleson in the psychoneuroses; at necropsy, Lewis showed that these patients have a small, aplastic circulatory system. Fulstow, somewhat later, published similar findings in a series of necropsies, though the percentage was not quite so high as that of Lewis. A correlation with the results of pathologic study is needed. The Schneider index is a test, and if it is used as such we can derive great value from it.

Dr. Israel Wechsler: I am in sympathy with the attempt of Dr. Huddleson to correlate physical manifestations with psychogenic symptoms, but I am afraid that the Schneider index is rather crude, not to say naïve. So many variables enter into the equation that nobody could correlate psychogenic manifestations with the simple facts presented. Dr. Huddleson speaks of 200 cases and of small percentages of this group. In thinking of psychoneuroses one does so commonly in terms of thousands of cases. How can one venture to make any statement on the examination of the figures in 200 varied instances? How can one venture to draw conclusions from them and hope to have the Schneider index taken seriously?

Variable factors entering into the problem are endocrine imbalance, toxic agents, disturbances of the vegetative nervous system and cardiac conditions, to mention only a few. Is not Dr. Huddleson putting the cart before the horse? Are the changes in pulse and blood pressure the result of the neuroses, are they symptoms of them, or are they linked with more fundamental alterations that

have nothing to do with the psychoneuroses? Every neurologist hopes that some day he will be able to speak of the psychoneuroses in terms of physiologic processes, but the Schneider index is hardly the fulfilment of that hope. At present, if one will spend ten minutes talking with a patient, he will get infinitely more information for diagnostic purposes than if he takes pulse and blood pressure readings. I have more than two thousand records of examinations of the pulse and blood pressure of psychoneurotic patients, which I once tried to correlate, but I found that I could draw no conclusions from them. All that one can say is that psychoneurotic patients have a labile pulse and blood pressure. While, therefore, the effort is justifiable, I think that it is better, at present, to speak of physiologic processes in terms of physiology, and of psychogenic processes in terms of psychology. One must examine thousands of patients, exclude or account for all variable factors and make absolute correlations before drawing any conclusions.

DR. JAMES H. HUDDLESON: Dr. Casamajor's definition of psychoneurosis and his criticism of our point of view is perfectly valid. We tried to eliminate complicated cases so far as possible, also cases in which there were other elements that might account for the low index or the disorder in circulatory efficiency. At the same time, we found certain cases that eventually were diagnosed by neurologists as psychoneuroses. Whether psychoneurosis is a state of mind or not, it is a disability of a sort. It is not necessarily the cause of circulatory dysfunction. We are not trying to show which is cause and which is effect. I do not feel that we are putting the cart before the horse. We wanted to show that with a psychoneurosis a cardiovascular system coexists that is working in a certain way, which can be measured to some extent by the Schneider index. We do not attempt to measure the psychoneurosis as such mathematically.

So far as the cases of gastritis and colitis are concerned, we believe, despite the small series, that, like the cardiac cases, they will also show a low Schneider index in a certain percentage of instances. For this reason, we believe that it would be better not to select the cases as yet. When enough are available, we may be able to present stronger statistics.

In reply to Dr. Hyslop's question relative to the effort syndrome, I might quote from the cardiologist, Wiggers, who says that the symptoms are pains in the chest, dyspnea, fatigue, giddiness, palpitation, precordial pain, tenderness and fainting attacks, in that order of frequency. I do not know whether that is exactly what Dr. Hyslop meant, but the cases of neurocirculatory asthenia are of this group.

In regard to Dr. Rosett's question as to whether we could say that everyone with an effort syndrome necessarily has a psychoneurosis, we do not think so. We have not examined the records from that angle; it should be done. We do not think that in every case in which myocardial or chronic valvular disabilities have been eliminated it would be possible to demonstrate psychogenesis.

Regarding Dr. Wechsler's comment, we wish to stress the same point that he brought up. In going through a large number of these cases, hundreds or thousands, we found that they did not give us any information. That fact appeared in the first part of our paper. No matter how many psychoneurotic patients one may examine simply for the diastolic and systolic pressure and the pulse rate, he will not secure any information about the psychoneuroses.

A CLINICAL CONSIDERATION OF MENTAL DEFICIENCY. DR. HOWARD W. POTTER.

I do not regard the term mental deficiency or the terms of any of its subdivisions, such as moron, imbecile or idiot, of clinical value. Mental deficiency, from the point of view of terminology belongs in the same category as the term insanity. A defect in intelligence is regarded as only one of the symptoms of a mentally deficient state. The functioning of intelligence may be interfered with because of structural defects, metabolic disorders or emotional conflicts.

In discussing the pathogenesis of an inadequately functioning intelligence, I find in one group of cases a generalized constitutional inferiority; in another group, infectious processes and trauma; in a third group, a peculiar degenerative or neoplastic-like process in the brain, and in a fourth group, a faulty emotional organization.

Knowledge as to neuropathology and neurohistology is inadequate. I wish to stress the clinical approach through constitutional, neurologic and psychiatric studies of persons belonging in the group of mental defectives.

DISCUSSION

DR. ALEXANDER N. BRONFENBRENNER (by invitation): We have just heard that the treatment and training of the feebleminded will be more effective when they are considered from the clinical point of view rather than from the point of view of mental age. Looking over the Proceedings of the American Association for the Study of the Feebleminded, one will notice that a similar idea has repeatedly been expressed. However, it never was emphasized as a step that should be taken immediately. The reason for hesitation on the part of colleagues interested in the problem of feeblemindedness has been derived from the peculiarities of the problem itself. Proper care of the feebleminded cannot be given in a private home or in an ordinary school or hospital. It requires a special institution, the organization of which should provide, in the first place, a proper home and a school; after this, a scientific study of the feebleminded should be made among the institutionalized patients. In presenting these cardinal requirements for the care of the feebleminded, I have merely reiterated the principles on which a foremost promoter of the cause of the care of the mentally deficient has built his now nearly completed institution.

The treatment and training of the feebleminded were arranged to fit the mental age level at a time when neither institutional organization nor the medical profession was in a position to undertake a scientific approach to the problem. However, the situation is changing. Dr. Potter recognizes these changes and acts accordingly. He has indicated the fields of medicine that are directly and mainly concerned with the morbid anatomy and pathogenesis of mental deficiency. The medical problems are hardly different from those in other fields of medicine; most of them are closely related to outstanding problems of neuropathology: developmental conditions of the brain, the advanced stages of various forms of encephalitis, the late results of birth traumas, damaged functional capacity of the brain as a result of premature closure of the skull, dysendocrinism in its extreme forms, etc. The brain of the feebleminded person might be considered as an experiment of nature providing an opportunity for the study of such cardinal problems as cerebral architecture, myelinization, localization, etc. The recent work of Hindze on the correlation between the richness of the vascular supply of the brain and mental capacity indicates another field in which the study of the brain of the feebleminded may be pursued. An opportunity for bridging the gap between degenerative and neoplastic processes, as illustrated by tuberous sclerosis, should be followed.

DR. GEORGE VAN NESS DEARBORN: We are indebted to Dr. Potter for enlarging the concept of feeblemindedness. However, I may suggest that psychologists have also attempted this. Constitutional psychopathic inferiority, that whole group of weaknesses of personality caused by the absence of the normal purposes, is so closely allied to feeblemindedness that the two concepts will sooner or later be shown to fuse into one another as "purpose lacks."

From his remarks, I am compelled to think that Dr. Potter slightly underestimates the practical importance of psychometry. Even if the present system of measurements were shown to be worth comparatively little, one could not do without it in routine work. Its procedure is open to improvement and development; no one knows that better than the psychologists who have spent so many hours in its use. Undoubtedly it is still used too mechanically and far too narrowly.

It seems to me that Dr. Potter also underestimates the advances that have been made in the neuropathology of feeblemindedness in the last few years. I

refer to the excellent, far-reaching contributions of the English neurologists, Tredgold, A. W. Campbell and S. J. Bolton, and to the work of D. H. Dolley, Mott, Craig, Watson, Herrick and Tredgold. Tredgold and J. A. Berry of Australia accept the point of view that the basal neuropathologic feature of amentia is a cytopenia of the upper layers of the cortex, the so-called neopallium (supragranular). One may well believe that in all probability this is the neuropathologic basis of feeblemindedness; it would seem likely that the upper layer of the cortex is in control of the lower, archipallic layers which represent instinct, habits and the other more automatic functions (behavior patterns).

DR. CLARENCE O. CHENEY: Dr. Potter's presentation has been constructive and far-reaching. I think that he has not made his remarks about intelligence tests from an academic or argumentative standpoint. From time to time we regret the emphasis that has been placed on intelligence quotients in questions regarding the training of children. I have particular reference to the determination of whether a child is placed in a special or ungraded class as decided by a certain intelligence quotient—I forget whether it is 75 or 76. If the child's quotient is one point above that arbitrary figure, he is kept in a regular class, where he may be a destructive influence; if it is one point below, according to the rules of the department of education, he may enter an ungraded class.

I hope that the conception which Dr. Potter has presented may be widely accepted and may perhaps eventually influence the attitude of educators toward the clinical conditions in the mental deficiencies. Dr. Potter has shown that these are not static conditions. Persons who at one time may have a poorly functioning intelligence change, as conditions are changed, in spite of what some psychologists have said in the past.

DR. HOWARD W. POTTER: I do not think that any clinical psychiatrist makes more use of mental tests than I do. My aim, however, is to go behind the mental tests and to arouse curiosity as to why a person has a condition of mental deficiency. I think that the answer to this must be sought for in terms of constitutional medicine, neurology or psychiatry.

MENTAL HYGIENE. A DEVELOPING CONCEPT. DR. GEORGE K. PRATT (by invitation).

In discussing mental hygiene as a developing concept, it is necessary at the outset to make a distinction between mental hygiene, the subject, with its various points of view and technic, and mental hygiene, the movement. Although they begin their careers as one and the same, the two are no longer always identical. The former continues to be fluid and changing; the latter is always exposed to the danger of crystallization at its present level. Neither can be or should be wholly separated from the influence of the other. But it is the various points of view and technic of mental hygiene that I wish to emphasize particularly in this paper.

When, twenty years ago, mental hygiene began as a simple reform movement to better the lot of the mentally defective in institutions, its roots were embedded in psychiatry, and psychiatrists properly directed and dominated its growth. It grew phenomenally. Soon its simple, original objective was expanded to include attention to the prevention of mental disease, and gradually the front lines of its efforts were pushed back from adult life to adolescence, from adolescence to childhood, and, more recently, from childhood to infancy. Each deeper thrust into these new territories required that mental hygiene evolve new funds of knowledge and new technics, some of which were completely foreign to orthodox psychiatry and particularly to that part of psychiatry which dealt with the end-results of mental disease—so-called institutional psychiatry. Thus, as mental hygiene's task of preventing mental disorder continued to grow, and as psychiatric conceptions of what constituted mental disorder broadened concurrently, the need arose for enlisting aid from other branches of medicine, as well as from scientific disciplines outside of medicine entirely.

As a result, mental hygiene acquired a new complexity; its points of view and its methods began to reflect doctrines other than those of psychiatry alone, for effective advance required that a liaison be established with the other technical resources in the community which experience had demonstrated were essential for preventive work. Necessarily, this new complexity of mental hygiene had to be accompanied by a corresponding measure of flexibility, if arrest of growth was to be avoided. Growth was not halted, however. Indeed, just the opposite proved to be the case. With the gradual development of an interpretative, dynamic psychiatry opening new vistas of understanding of the wellsprings of human behavior, the increased opportunities for mental hygiene's task of prevention threatened to overwhelm those engaged in the work.

Today the concept of mental hygiene is, in every sense of the word, a developing one. It has broken with those whose limited vision would have kept it a static concept; it is no longer even an exclusively psychiatric concept, unless one concedes the validity of a difference between the concepts of conventional psychiatry on the one hand and a socialized community psychiatry on the other. Theoretically, there should be no difference between these concepts; actually, everyone recognizes that such a difference sometimes exists. In brief, mental hygiene has developed and continues to develop in a fashion comparable to the emotional growth of the child who begins life under the constant supervision of the parents, and who is wholly dependent on them. As emotional growth continues, however, the child is increasingly subjected to socializing influences from outside the family circle. His personality reflects more and more the attitudes of other people, and he tends, in adolescence, to incorporate into his philosophy and behavior some of the points of view and ideas of the socializing influences that have impinged on him. With the attainment of adult life, he emancipates himself from the state of utter dependency on his parents. Nevertheless, in the flush of his new freedom and broader development, he still recognizes his organic relationship to his parents, and his matured affection for them and his loyalty to them remain intact.

This is what has been happening to mental hygiene and its parent, psychiatry. Stemming originally from psychiatry, mental hygiene has developed, not along parallel lines, but along gradually divergent ones. Today mental hygiene is still rooted in psychiatry, but its field of activity has grown to be more inclusive than that of psychiatry.

To illustrate what I mean, let me ask you to visualize a horizontal line representing the development of mental hygiene. Let the left extremity of the line stand for mental hygiene as it was in its simple beginning. Presently, as one travels toward the right, one sees the contributions of other bodies of knowledge finding a place along this line. Here is neurology, with its special point of view. A little later, internal medicine makes its appearance. Pediatrics soon joins the others, to reenforce the medical concern with the subject. Somewhere toward the end of the first third of the line, the development of the concept is further strengthened by the entrance of nonmedical disciplines. Thus, psychology, sociology, jurisprudence and theology all find places in the total concept. Education, with its particular pedagogic philosophies and methods, occupies a strategic position on this imaginary line, while social case-work is far from being the least important of the contributions.

Along this line, then, are rightful, needful places for many scientific disciplines and bodies of knowledge. Here is a concept of mental hygiene as a hybrid, a composite, even a synthetic, creation, made up of a number of ingredients, which, when properly integrated into a common pattern, become effective for a better understanding and management of human behavior. This illustration also justifies the view of mental hygiene as a developing concept; the individual components of mental hygiene are themselves continually in a state of flux. Research in internal medicine, for example, is continuously throwing new light on the relationship between soma and psyche, thus requiring frequent changes in point of view and methods of treatment. Advances in the field of pediatrics are taking into account more and more emotional relationships between children and parents as explana-

tions for certain pediatric problems. Education is scrutinizing with new objectivity the wholesomeness of pedagogic practices when viewed from the angle of the adjustment of pupils. Even law, rooted in archaic precedents as it is, shows an encouraging, if ponderous, interest in a few of the more dynamic motivations for unusual behavior.

With each new advance in knowledge on the part of any one of its components, mental hygiene, the total concept, must be sufficiently flexible to permit of a rearrangement of its points of view and technic in order to utilize constructively the new accretion of knowledge. For this reason, any delineation of its form and outline can be valid only for a given moment.

But while mental hygiene has enlisted in its composition the contributions of many other scientific disciplines, it has not been wholly selfish; it has also given in return. One need only point to the influence of its point of view in the philosophies and in the methods of social case-work, of progressive education, even of some of the fundamentals of medicine itself, to realize how thoroughly this infiltrative process has been carried out, and how drastically, in some instances, it has transformed and reinvigorated former attitudes that had all but reached the end of a useful phase of their growth.

This rate of infiltration has not been uniform. Even at its present stage of development, mental hygiene has made scant progress in some directions. A case in point is that of sensitization of students in theological seminaries to a more effective insight and understanding of human nature in trouble. Another is that of acquision by medical students of a point of view that links numerous maladjustments in adult life with faulty training in childhood by the parents. Still a third example of an as yet unutilized opportunity for the further infiltration of mental hygiene into other bodies of knowledge is found in parent education. Because this example is typical and because it is one that demands serious attention, I shall digress for a few moments to elaborate on it.

From the time when mental hygiene added to its original aim of promoting the welfare of the mentally defective in institutions a concern for the prevention of mental disease, the need for getting parents to adopt certain attitudes toward their children and to abandon others has been recognized. Indeed, one of the earliest maxims in mental hygiene was: "You can't change the child's undesirable behavior until you first change the parental attitude which caused that behavior."

In its early years, much of the educational effort of mental hygiene was expended in lecture courses the purpose of which was to give information to parents, in the belief that, after being told which attitudes were health-provoking, the parents would at once divest themselves of unhealthy ones and proceed to display instead those extolled by the lecturer. Incidentally, most of the instruction in those pioneer days was limited to telling parents and teachers what to avoid. But as this educational work continued, it became apparent that it was one thing to tell a parent what he should do in the matter of child training and quite another to persuade him to apply dutifully the information that he had received.

To overcome this difficulty, mental hygiene turned once more to the source whence so much of its strength was beginning to come, i. e., the fountainhead of a dynamic, analytic psychiatry, and thereon discovered the obvious. Many parents, consciously honest and sincere in their desire to exchange attitudes that adversely affected the behavior and mental health of their children for more wholesome ones, were, nevertheless, not psychologically free to do so; they were in the grip of unconscious forces stronger than their conscious desires. How to free psychologically these parents so that healthy attitudes could be displayed, with resulting benefit to their children became the problem confronting mental hygiene. An emancipating procedure, consisting of individual psychiatric treatment of the hundreds of thousands of parents making up the audiences of lecture courses and child study groups throughout the country, was obviously impossible. So it was necessary to enlist the aid of modern psychiatry in devising methods of teaching parents in mass groups, not only to give them an understanding of the significance of their attitudes in relation to mental hygiene, but, infinitely more important,

to get them to accept emotionally and to digest the resistance-arousing interpretations, so as to produce self-insight, as a necessary prelude to changes in their attitudes.

While a beginning has been made in attacking this problem, progress is slow. In the meantime, a laymen's creation known as the parent education movement has been growing independently and phenomenally. Its objective is the better education of parents in all aspects of child care, physical and recreational, as well as psychologic. Hundreds of parent education study groups, usually with no participation by the professions of medicine, psychiatry or mental hygiene, are being conducted in all parts of the country. But the responsible leaders of the parent education movement have begun to realize that their methods have reached a point beyond which no progress is being made, that the mere imparting of information to parents is not always enough to insure its proper application. And some of them, at least, are turning to mental hygiene for aid in working out the solution of their common problem. In this particular instance, mental hygiene has much to offer and much to receive. Many of its points of view can with profit be infiltrated into the parent education movement; at the same time there are many technics of the latter which mental hygiene needs to borrow.

Another reason for the lack of uniformity in the rate of mental hygiene's general infiltrative process is found in its dependency on the progress already attained in psychiatry. Psychiatry is in transition—transition from the epoch-making description and classification of a half century or more ago to the dynamism and interpretation just getting well under way. At the moment, divergent views befog the issue. Defenders of the traditionally established era remain in power in most places and continue to color the subject within their sphere of influence according to the views with which they are familiar. They are reluctant to relinquish a long and eminent leadership to champions of the new whose strength and leadership have not yet been completely tested. So progress in psychiatry, caught between two fires, must advance less vigorously than might otherwise be the case, until the period of transition is ended and a new era of mutual agreement and singleness of purpose is established.

Mental hygiene, the subject, and particularly mental hygiene, the movement, are bound to be affected by the current divergence of psychiatric views. A crack has already appeared in the integrity of the composition of these two entities of mental hygiene. Whether the crack will be permitted to widen to a chasm, or whether entrenched reactionism on the one hand, and rebellious liberalism on the other, will succeed in adjusting their differences on a basis of mutual toleration and common striving, is a problem the solution of which hangs momentarily in the balance.

It is encouraging to note, however, that no one body of scientific knowledge is monopolistically represented in the concept of mental hygiene. But this may not always be true, and further development inevitably will be retarded if the old fallacy is perpetuated that mental hygiene and psychiatry are synonymous. These terms have been used interchangeably in the past. In the beginning there may have been an excuse for this, since the mental hygiene of a quarter of a century ago was so closely identified with psychiatric efforts to cure or prevent mental disease as to be indistinguishable from them. I would like to submit that mental hygiene today, despite a loyal recognition of its inseparable connection with psychiatry and of its fundamental dependency on the principles and technics of psychiatry, is nevertheless a broader, a more inclusive and a different concept. Psychiatry, whether it is the orthodox variety or a liberalized modern social psychiatry, is that branch of medicine concerned with the treatment and prevention of mental disease. On the other hand, mental hygiene has been tending unmistakably in the last year or two toward a primary concern for mental health. I need not emphasize the complete and fundamental differences in these two objectives. Up to the present, virtually everyone has accepted as the ultimate destiny of mental hygiene the prevention of mental disease. Such, originally, was also the aim of preventive medicine, and all are familiar with the development within the field of

preventive medicine which is causing a growing portion of its efforts to be dedicated to the promotion of health, rather than to the prevention of sickness. Mental hygiene is traveling in a parallel direction so rapidly that interest in this next phase of development on the part of mental hygiene, the subject, has already outstripped a similar interest manifested by mental hygiene, the movement.

Once accomplished, this significant shift in emphasis from disease to health promises to lead mental hygiene into still more constructive fields of endeavor. One anticipates, for example, the formulation in usable shape of certain principles of mental health, with suitable methods of application to the problems of health education as taught in the public schools. At present, health education in the schools, its principles and technic, consists exclusively in teaching the child facts about the building up and conservation of physical health. I have been unsuccessful in discovering a single health education program in the United States in which an adequate quality of instruction in mental health plays a part. To be sure, attempts to give such instructions have been made from time to time (usually by teachers of health education and not by competent mental hygienists), but in almost every instance the efforts have failed to evolve anything more practical than a set of sterile platitudes. Such fruitless results are inevitable if the establishment of habits of mental health is approached from the angle of the traditional concept of mental disease.

In order to effect a shift in emphasis, mental hygiene must establish new relationships and develop new methods. The building up of sound, positive mental health will make new demands on psychiatric knowledge, as well as on facilities for research in a variety of other fields. Fascinating problems will challenge to the utmost the ingenuity of the investigator. What, for example, are the conditions necessary for a healthy mental life? What, psychologically speaking, do human beings require in life? What constitutes healthy parental attitudes toward children, and how may these attitudes be developed? These and a host of other, as yet unanswered, questions constitute the field of opportunity for the mental hygiene of the future. In their solution will arise, once more, the need for mental hygiene to continue as a developing, ever-changing concept, recruiting to its service the forces of many other scientific bodies of knowledge and pressing toward its goal of liaison agent which coordinates every pertinent fragment of knowledge concerning human behavior and the methods of shaping this into sturdy mental health. The personnel for these tasks is lacking or undiscovered. Programs of training will have to be instituted under the direction of a few pioneers of vision and ability. Increasing pressure will have to be brought by mental hygiene, the subject, on mental hygiene, the movement, to overcome traditional modes of thinking and that indisposition to change which characterizes conservatism, wherever found. But these obstacles are not insurmountable. Mental hygiene, the subject, has faced them before, and under the aegis of leadership drawn largely from the ranks of a progressive social psychiatry, mental hygiene may be expected to advance into new fields of usefulness.

DISCUSSION

DR. LAWSON G. LOWREY: I shall discuss only certain points which I wish to emphasize. I draw directly from clinical experience of the past ten years in the study and treatment of children presenting problems of personality and behavior. The work has been done under the most difficult circumstances it is possible to imagine, namely, while the child is in his ordinary environment, surrounded by all the influences, often pernicious, which have had a part in bringing about his difficulties. The clinical groups, as originally set up, were based on Southard's notion of a psychiatric team: the psychiatrist, psychologist and psychiatric social worker, coordinated into a cooperating team. Today this is a commonplace, but then such organization had not been worked out well, and there were many difficulties involved in developing the needed cooperation between people trained in such different disciplines.

In the course of the years practically every known type of case has been seen. From the standpoint of constitutional make-up and disease, one finds, of course, all sorts of neurologic conditions and all kinds of organic and functional states in children of different ages. There are children in need of pediatric care, of surgical attention and of all varieties of special medical treatment. Many children present none of these physical problems, none of the deviations, constitutional or acquired, which are often regarded as being responsible for the problems of personality and behavior. The children are seen in the midst of their struggle for psychologic adequacy and stability, involved in all sorts of complicated emotional relationships in the home, where parental jealousies and conflicts, jealousies among the children, etc., are threatening them in their struggle. In every case, whatever the therapeutic approach may be (physical, psychologic, etc), some type of environmental problem must be met. I have been accustomed to speak of change of environment as involving two kinds of manipulation. On the one hand, there is the manipulation of things, such as getting the child into a different school, into recreational groups, or out of the home into an institution or foster home. On the other hand, and much more important, there is the manipulation of people; here all sorts of direct and indirect methods must be used. In connection with this need for environmental change, it seems to me that the distinction between psychiatric work and social psychiatric work (or clinical mental hygiene) lies in the necessarily more extensive manipulation of the environment by the latter than by the former. In molding the environment, the psychiatrist has some experiences for which he has not been prepared in his formal training. He must understand and utilize community resources of many types in trying to correct the problems with which he is confronted. There is need for various types of programs in the community, which must be coordinated with the work of the clinic. The complexity of all this in straight clinical work reenforces Dr. Pratt's point that the concept of mental hygiene is a developing one and not one completely crystallized.

As I see it mental hygiene is not solely the job of any one group in the community, although leadership is clearly to be expected from some group, and the psychiatrists have the most to contribute to such leadership. The mental hygienists have been vigorously criticized because they did not include educators and others in their clinical teams, and sometimes it has been difficult to answer the criticisms. It has been amusing to see which groups criticize the mental hygienists, because it has often happened that those who offered the most criticism had, after all, nothing to contribute. Certainly the psychiatrist has the most to give in pointing out the direction in which the whole program must go, on the basis of his clinical work and in terms of complete organization for dealing with clinical problems. Some real educational problems have developed, not only for the medical profession, but for all those professional groups which have to do with children in large numbers. There are also the parents. There is need for a sound, well rounded presentation of the facts at hand before all these groups, in order that they may, in their daily contacts, contribute to the mental health of their charges rather than to their mental ill health.

Dr. George S. Stevenson (by invitation): The genesis of mental hygiene, as outlined by Dr. Pratt, makes it possible for one to think of it as existing at four levels—the custodial, the therapeutic, the preventive and the positive. Because growth has been from the custodial toward the positive level, one may be inclined at times to look back on and question the legitimacy of the earlier aspects. However, the only safe attitude to take is that which permits each of these four aspects to be expressed in its appropriate place. The acceptance of a positive mental hygiene does not carry with it the rejection of the custodial, therapeutic or preventive aspects. Not infrequently, however, this rejection occurs, and Dr. Pratt's analogy of the relationship between the development of mental hygiene and the development of the child may, on the basis of identical human motivations, be considered a homology, since it not infrequently happens that someone identified with the maturing mental hygiene movement rejects the parent psychiatry and tries to get along without it; similarly we find an overprotectiveness on the part of the parent psychiatry, which at times warns its "child" against association with nonmedical companions.

One of the difficulties is, I believe, the failure to understand what is meant by the term positive mental hygiene. Actually, the concept of positive mental health should be easier to understand than the positive in any other aspect of health, since all the mental hygienist's concepts of enrichment and satisfaction are in terms of the whole human being rather than of his organs. Positive mental health merely means the development of potentialities for a fuller experience, with greater satisfaction and social constructiveness. The endeavors that influence this development necessarily touch on every aspect of human activity, and it is this breadth of effort that frightens off some who would attempt to understand what is meant by positive mental health. But being a mental hygienist in the positive sense does not imply the encompassment of all the details of all the fields of service; it merely implies seeing the implications in the fields so as to be able to lead those who are in more complete grasp of the details to a common goal of human satisfaction and productivity.

It must be evident, therefore, that, with the adoption of the concept of positive mental health, the artificial barriers within the community such as departments of health, education, welfare, etc., dissolve somewhat, as do also those of academic life; the resulting contact of otherwise sharply demarcated fields is producing combinations of effort and understanding that are developing new procedures. One finds social service used in private practice; problems of human motivation and social determinants are being reorganized in the art of medicine and are considered proper subjects for dignified scientific study. Again, recreation is drawn into the province of delinquency, and psychiatric contributions are applied to social case work. A positive concept of mental health should create dissatisfaction with objectives and technics as they stand.

Dr. Pratt also has called attention to the fact that the teaching of mental health in schools and colleges has resulted in merely the laying down of a few platitudes. If the regular subjects of the current curriculums of schools and colleges have to do with the future satisfaction and productivity of the students (and what other excuse is there for them?), is it not fair to question whether mental health can be taught as a thing apart from these subjects, whose aims are supposedly those of mental health, and is it not proper to assume that the best teaching of these subjects will result in as good instruction in mental health as we can hope for?

In carrying over this principle into other fields, the best kind of mental hygiene in nursing, theology, law, social service and medicine is merely nursing, theology, law, social service and medicine perhaps devoid of any label of mental hygiene, but so constructed as to function at the highest level in meeting human needs.

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

TRACY J. PUTNAM, M.D., *Secretary*

Jan. 12, 1933

EDWARD S. ABBOT, M. D., *President, in the Chair*

CENTRIFUGAL INFLUENCE ON CENTRIPETAL SYSTEMS IN THE BRAIN. PROF. BERNARDUS BROUWER, M.D., Amsterdam, Netherlands (by invitation).

My interest in the problem of the centrifugal influence on centripetal systems in the brain was evoked during investigations on the cerebral optic system. After having studied the projections of various parts of the retina on the calcarine cortex, my associates and I found that many fibers originating in the area striata return again to the external geniculate body. We made lesions in various parts

of the calcarine cortex in rabbits and in monkeys; the animals were killed after eighteen days, and the secondary degenerations were studied with the help of the Marchi method. For many years it has been known that several fibers proceed to the anterior colliculus, which is a highly developed reflex center, especially for the movements of the eyeballs and of the pupils. We were not the first to show that several fibers also proceed to the external geniculate body itself. Probst, Beck and Bouman had already seen these in lower mammals, but in their day it was not possible to connect this anatomic fact with physiology. Biemond worked out this problem in my laboratory, especially in monkeys. The fibers proceed in the central optic radiation but have a distinct tendency to remain in the inner layer, called the internal striatum, while the centripetal systems prefer the outer layer, called the external striatum. Biemond's studies showed that there is a distinct localization between the calcarine cortex and the external geniculate body, corresponding with that of the centripetal systems. The frontal part of the calcarine cortex is connected with the frontal part of the external geniculate body, the caudal part with the caudal part of the geniculate body, and so on. Hence this relation is the same in both directions. What may be the significance of such descending tracts? The idea at once arises that the external geniculate body is a reflex station, and that the calcarine cortex may exercise an influence by such descending tracts on the optic reflex center, just as it does on the anterior colliculus. It is possible to defend this idea because investigators believe that the external geniculate body is connected by special fibers with the anterior colliculus. In the external geniculate body of monkeys—and also of man—there are some layers of bigger cells in the ventral part of the ganglion. In studying the retrograde degenerations in the external geniculate body in cases of lesions in the calcarine cortex in man, many investigators have found that the larger cells do not disappear. Hence the conclusion has been drawn that these cells do not send their axis cylinders to the calcarine cortex, but to lower centers in the nervous system. In this way the ganglion could be regarded as a highly developed optic reflex station. My own experience goes in a different line. I saw degenerations in the layers of large cells in several old lesions of the calcarine cortex in man. Furthermore, my associates and I have made extensive lesions in the calcarine cortex in monkeys and have found that these cells also undergo complete degeneration provided the animals live for a long time after the operation. I became convinced that the large cells degenerate somewhat more slowly than the smaller ones. This may explain the different results of other investigators.

Hence, it is impossible to regard the external geniculate body as an ordinary reflex station, because the centrifugal fibers to the motor centers are lacking. The descending fibers of the cortex here touch cells that receive their impulses directly from the retina. We believed that this fact may be of significance for physiologic deductions.

To control this relation in other sensory regions I started investigations with Posthumus Meyjes on the acoustic area. The acoustic fibers enter the brain in the cochlear nerve and are interrupted in the medulla oblongata chiefly at two centers, the tuberculum acusticum and the ventral cochlear nucleus. From these nuclei long tracts proceed to the posterior colliculus and to the internal geniculate body. This ganglion is connected by many corticopetal fibers with the temporal lobe, the highest center for acoustic impulses. If this cortical area is removed in animals, a retrograde degeneration in the internal geniculate body is evoked. But our investigations proved that the findings of d'Hollander were correct, namely, that there are also many descending tracts returning to the internal geniculate body. We found even that there is localization in this respect, that special parts of the temporal lobes are related to special parts of the internal geniculate body. While this ganglion does not send fibers to motor centers in the nervous system, there are the same relations here as in the optic mechanisms. The cortex may have an influence on stations having a purely sensory character.

A third region is the optic thalamus. This is a complicated part of the central nervous system; hence I must give a fuller demonstration of its relations. There are two chief parts in the optic thalamus, an older and a newer one, the paleothalamus and the neothalamus. The phylogenetic development of the optic thalamus,

after Clark, is as follows: In the lowest vertebrates, for example, the cyclostomes, only the hypothalamus and the epithalamus are present. Furthermore, some cell groups surround the ventricle, and are called the periventricular system. The visceral tracts originating in lower parts of the nervous system and some poorly developed tracts for other sensory functions terminate in the hypothalamus. The epithalamus, the so-called habenula, is connected with olfactory functions. In reptiles, the neothalamus develops but is still small. One can distinguish here an anterior, a medial and a small ventral nucleus. Several tracts conducting sensory stimuli, as pain, temperature, tactile and proprioceptive impulses, terminate in the ventral and in the medial nuclei. In reptiles there is practically no neopallium; hence one cannot speak here of thalamocortical connections. These sensory fibers, however, are connected with the striate body, and in this way some higher movements are made possible. In the ascending scale the neothalamus develops further and gradually dominates the paleothalamus. In lower mammals, the nuclei of the neothalamus are still small but develop more and more and are largest in man. The ventral, anterior and medial nuclei of primates and man are already present in reptiles, but they are much larger in the former. There are several subdivisions of these nuclei. The sensory fibers, conducting proprioceptive impulses and the various qualities of cutaneous sensibility coming from below, terminate chiefly in the ventral, but also in the medial nucleus. This is the case not only in the extremities and the trunk but also in the various sensory functions of the head. There is here, however, one big nucleus, which is not present in lower animals; it appears in the scale of mammals and gradually gets larger in the primates; that is, the lateral nucleus. All these nuclei are projected on the cerebral cortex, and in this way, among other things, conscious sensations are made possible.

Much work has been done to analyze on what part of the cortex each nucleus is projected, but opinions differ greatly. Some time ago, with Dr. Sager from Bucharest, I began a new series of investigations on this subject. We started from the conception published by Dusser de Barenne on the localization of the sensory area on the cortex in monkeys. He put strychnine on the cortex and saw symptoms of sensory excitation (hypersensitivity of the skin and deeper structures). These symptoms appeared in different parts of the body according to the cortical areas strychninized. There is a sharp distinction between the areas for the leg, arm and face. This sensory area is much more extensive than was expected, but, in my opinion, this is the best conception yet known. Poljak, of Chicago, after many extirpations of the cerebral cortex and much study of the degeneration in the optic thalamus, came to conclusions quite near to those drawn from the experiments with strychnine. Dusser de Barenne removed small parts of the sensory cortical center of the face, arm and leg and also of the frontal area. The animals were killed after five months; complete serial sections were cut in my laboratory and stained with the methods of van Gieson and Weigert-Pal. Retrograde degeneration of many cells and fibers was present in the various nuclei of the optic thalamus. We could study here the projection of the sensory area of the face, arm and leg, and also the frontal region.

The chief results (illustrated by glass models in which it is possible to study the projections in three dimensions) are: The area of the face is situated more caudal than that of the arm, which is somewhat more frontal, while the leg is the most dorsally projected. The frontal area is projected on the medial and lateral nuclei. There is, however, an extensive overlap. This means that there is only a rough localization of the various parts of the body in the optic thalamus. From the anatomic side, then, there are striking facts of significance for the problem of a topographic localization. Many sensory fibers coming from below terminate in the caudal part of the optic thalamus; for example, those from the legs; none of them reach the lateral nuclei directly; still I have found that this latter nucleus is immediately connected with the sensory cortical area. Hence I reach the conception that there is a double projection of these systems on the neopallium in higher mammals. One of these is formed by association tracts between the ventral and medial nuclei, on the one hand, and the dorsolateral nuclei on the other. The optic thalamus may work as a sensory *unit*. There is a regrouping of the sensory

pathways in the optic thalamus; but furthermore there must be also a regrouping of these sensory pathways between the optic thalamus and the cortex, so that a part of the impulses issuing from various parts of the body may be conducted to separated parts of the cortex. A topographic localization of the sensory systems for the arm, leg and face is prepared in the optic thalamus, but is finally perfected in the neopallium.

A remarkable fact is that many fibers, descending from various parts of the cerebral cortex, return to all the nuclei of the thalamus. This is proved by the extirpation of parts of the cerebral cortex and by study of the secondary degenerations with the help of the Marchi method. This had been shown earlier by d'Hollander, Poljak and others, but there are doubts about the results. Therefore I repeated the investigations, and I am sure that the number of the descending fibers in rabbits is enormous, and that they are present also in monkeys. They proceed to all the nuclei of the optic thalamus, except to the so-called periventricular system surrounding the midline. This fact is identical with what was seen in the optic and acoustic regions. Here, however, the problem is somewhat more complicated, because the thalamic nuclei are also connected with the striate body, which bears a motor character. On the other hand, it is sure that the principal and dominating function of the optic thalamus is that it forms a highly developed station for the elaboration of sensory stimuli.

Finally, I may say something about the termination of the pyramidal tracts. It is often maintained that the corticospinal tracts terminate exclusively around the motor cells of the anterior horns of the spinal cord. This is not correct, as von Monakow, for example, pointed out. He found that these fibers terminate in the basal part of the posterior horns and in the lateral horns, the gray substance lying between the posterior and anterior horns; Ariëns Kappers has emphasized the significance of this fact. The expression "motor tract" must be understood *cum grano salis*, because in most cases descending tracts do not end in motor nuclei but nearly always in coordinative centers. This, in his opinion, is strikingly illustrated by the fact that the dorsolateral pyramidal tract does not originally descend in the lateral but in the posterior funiculi of the spinal cord. This is so in marsupials, ungulates and several rodents. In carnivora and primates they run in the lateral funiculi, near the posterior sensory horns, and do not acquire direct relations with the motor cells themselves. I agree fully with this opinion in the case of rabbits; in the lantern slides shown it is seen clearly, with the Marchi method, that the pyramidal tracts in rabbits end, in the cervical part of the spinal cord, not in the ventral horns but at the base of the posterior horn, and in the region between this and the ventral horn. As yet it is not known where the uncrossed pyramidal tract, descending in the anterior column, terminates. The position of the tract suggests a direct termination in the ventral horn. In this connection it is important to state that probably these fibers influence the musculature of the trunk.

A totally different condition is seen in the spinospinal connections. In the case of a lesion in the spinal cord itself, it is striking that such short fibers—connecting the various levels—enter directly the gray substance of the anterior horns. These are all pure reflex systems. The same is the case with the deiterospinal tract, serving cerebellospinal reflex actions. Probst showed that these fibers terminate immediately around the motor cells of the anterior horns. There is some difference of opinion regarding the termination of the rubrospinal tract. Its position in the lateral columns near the posterior horns suggests that this tract does not end around the motor cells. Spitzler and Karplus found, indeed, that they reach only the region of the gray substance lying between the anterior and ventral horns. It cannot be an accident that this tract terminates in the same way as the pyramidal tracts. In higher mammals the nucleus ruber is, through seyeral tracts, intensively influenced by the cortex. Much work is still to be done concerning the finer endings of these so-called motor tracts, but it seems to me that the following idea may be presented: the descending tracts serving for pure reflex movements send their stimuli immediately to the motor cells; the descending tracts, however, that stand under the direct influence of the extensive association mechanism of the cortex send their impulses chiefly to the sensory and associative parts of the reflex arcs.

They do not all immediately charge the motor cells but have an influence in the reflex arcs, especially in their sensory part.

In summary, it may be stated that several descending systems influence the sensory side of the reflex arcs, and many terminate in centers that are of a purely sensory character. In my studies on the cerebral organization of vision I laid stress on the centrifugal side of the elaboration of the sensory functions. I did not know at that moment that Wallenberg, in Germany, had already written of theoretical conceptions closely resembling mine.

The corticofugal systems descending to the thalamus may not only regulate striatal movements, but must also have an influence on purely sensory functions. Some physiologic experiments prove that this is correct. In his experiments with strychninization of the sensorimotor area in the cortex of monkeys, Dusser de Barenne mentioned that the hyperexcitability in the limbs of both sides caused by the strychninization is still present in both forelimbs after extirpation of the whole sensory arm area of the opposite hemisphere and also of the greater part of the arm area of the hemisphere homolateral to the poisoning. The only differences in the reactions of the animal before and after the extirpations of the cortex was that, whereas the animal localized the stimuli with great promptness and reacted with sharp and very accurate movements of defense, this was no longer so after the cortical lesions. Dusser de Barenne believed that by strychninization not only the cortical area but also other nervous structures in other parts are set on fire. He thought, in this connection, in the first place of the thalamus opticus which in its ventral and ventrolateral nuclei represents, as is already known, one of the most important and functionally most highly developed subcortical sensory centers.

It is clear that the anatomic facts regarding the descending corticothalamic tracts support this point of view. By strychninization, also, the cells of these tracts are stimulated, and hence the increased activity could be transferred to the thalamus opticus. There are many association fibers between all these nuclei which may transfer the stimuli to the crossed hemisphere. This proves that such a centrifugal influence on sensory centers must be of great significance to physiology, and I share the opinion of Wallenberg that knowledge of these centrifugal connections must also have value for psychologic deductions. In the psychologic process which is called attention, some nerve cells have to be activated and others have to be inhibited, because undesirable stimuli must be prevented from entering consciousness. For example, in the optic regions one may conclude that during the state of attention some nerve cells of the external geniculate body may be stimulated; others, inhibited. The dominating influence of the cerebral cortex determines which stimuli may enter consciousness. There is no reason to deny that such an influence should go further in the lower sensory systems. Hence one accepts that there is also a centrifugal side in the process of sensation, of vision, of hearing and so on. I believe that a further analysis of these descending tracts to pure sensory centers will also help physiologists and psychologists to understand some of their experiences.

PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting, Jan. 27, 1933

A. M. ORNSTEEN, M.D., *President in the Chair*

FAMILIAL MUSCULAR ATROPHY. DR. ALFRED GORDON.

CASE 1.—Mrs. S. G., aged 57, the mother of two sons, began at an early age to observe a gradually developing weakness in the upper part of her right arm. At the same time she noticed a slight difficulty in walking. Both conditions gradually increased in intensity. Several examinations made within the last four months

revealed inability to raise the right arm; when an attempt is made to elevate it at even a slight acute angle the trunk leans backward. The power of the individual parts of the arm is greatly reduced, and there is a slight atrophy in the upper part. The musculature is flabby. The fingers of the right hand assume a claw-like position when the patient attempts to extend them. The pectoralis area of the right side of the thorax is decidedly flattened, especially in the subclavicular and supraclavicular regions. The dorsal region shows a strikingly receding right scapula and marked scoliosis, with lordosis in the lumbar region. The latter is evidently of a compensatory nature; in order to maintain the equilibrium of the body the trunk leans backward and the abdomen protrudes forward. About one year ago the patient also began to experience a slight difficulty in raising the left arm, but there is no conspicuous atrophy such as is observed on the right side, although the musculature is flabby. The tendon reflexes are not obtainable in the upper part of the right arm; a triceps reflex is elicited on the left side only, althought it is much diminished. There is no visible atrophy in the lower extremities. The face shows a glossy smoothness, the skin appears stretched, and there is a slight deviation of the face to the right. The orbiculares palpebrarum do not contract to their full extent. The emotional expression of the face is incomplete; the first impression is that of a parkinsonian facies. Evidently the atrophy is spreading to the musculature of the face. Electrical reactions show a diminution of faradic and galvanic response, but there is no reaction of degeneration.

The history reveals that a similar condition was present in the patient's father; it is seen also in her two sons. This is, therefore, familial dystrophy of the facio-scapulohumeral form.

In addition to the picture described, other anomalies of a grave character are found in the lower extremities. The gait is not ataxic, but the patient walks with the legs widely separated, and a distinct steppage is strikingly evident. Bilateral drop-foot is present. The extensor groups of the ankle muscle are paralyzed on both sides. The next important symptom is the total loss of the tendon reflexes: the patellar, achilles, abdominal and masseter reflexes are not obtained even with reenforcement on either side. The plantar reflex is variable; at times one sees flexion and at times extension of the toes, with different methods of testing. There is more frequently an extensor reflex on the left side. There is no paralysis of the extremities, and sensations are normal. There is no pain. The legs are somewhat cyanosed and cold. The feet are in the form of pronounced valgus; there are corns and calluses on the inner side of the soles. Inspection shows a constant dorsal extension of the left great toe, such as is seen in Friedreich's ataxia. As to the sphincters, the patient claims to have had imperative micturition with occasional incontinence for several years, but she also states that she had had catheterization of the ureter done by a surgeon, so that it is difficult to determine with accuracy the nature of the sphincteric disorder.

Case 2.—J. G., aged 27, the son of the patient in case 1, an upholsterer, presents a muscular atrophy similar to that of the mother. The onset occurred in childhood. A few years ago he began to experience difficulty in raising the right arm. The atrophy is less pronounced than that of the mother. He cannot raise the right arm gradually but only with a sudden and abrupt jerk. The flexor group of the right arm is flabby. The biceps and triceps reflexes on the right side are much diminished; the right scapula recedes markedly from the dorsum. The flattening of the pectoralis area and of the supraclavicular and subclavicular regions is evident. The lower extremities are normal.

Comment.—These two patients present an identical disorder in the musculature of the right scapulohumeral and upper dorsal areas, which is more pronounced in the mother. The mother, as well as another son and her own father, present in addition to the identical atrophic state a widespread areflexia, steppage gait, bilateral drop-foot and valgus and a Friedreich's great toe on one side.

These cases are examples of inherited malformations of several systems, namely, the central nervous system, the osseous system of the feet and the muscular apparatus. They probably belong to the intermediary or transitional forms between Friedreich's disease and other familial abnormalities.

DISCUSSION

DR. JOSEPH C. YASKIN: Does not muscular dystrophy of several years' duration usually produce an absence of reflexes? What evidence is there that in this case there is an involvement of the nervous system? Are the biceps and triceps reflexes preserved?

DR. GORDON: There is a slight triceps reflex but no biceps reflex. Atrophy is confined to the upper part of the body. The mother has marked lordosis, with some scoliosis, as well as muscular atrophy in the shoulder.

TONIC EPILEPTIC ATTACKS PRECIPITATED BY FRIGHT OR SURPRISE. DR. MELBOURNE J. COOPER.

The occurrence of epileptic seizures or variants of epileptic attacks caused by psychic trauma in some form has been recorded many times. Cobb (ARCH. NEUROL. & PSYCHIAT. **27**:1245 [May] 1932) mentioned the case of a boy who, having been frightened by dogs on former occasions, was thrown into a fit when a dog in the laboratory barked. Holmes (*Brain* **50**:720 [Oct.] 1927) described a case in which sudden falling without loss of consciousness, a manifestation that was regarded as epileptic, could be brought about by incidents causing surprise or by noise. He mentioned, as examples of such precipitating factors, a cat jumping on a table, an unexpected touch from behind and the slamming of a door. C. P. Symonds, in discussing Holmes's case, told of his own observation of a case in which the sound of a musical instrument would precipitate an epileptic attack. Foerster (*Lancet* **2**:309 [Aug. 8] 1931) noted that in a few persons with lesions at about the middle or posterior part of the first temporal convolution (presumably on the left side of the brain in right-handed persons) attacks were produced by sudden sharp noises.

The patient whom I present has generalized tonic seizures which occur spontaneously or as the result of incidents that surprise or frighten him. When the patient was first seen in Dr. Spiller's service in January, 1932, his father demonstrated a method by which an attack could be precipitated. Standing behind the patient he flicked his finger unexpectedly against the posterior surface of the pinna of the patient's ear. Immediately a tonic attack of the usual type occurred.

History.—This Italian man, aged 27, is a patient in the service of Dr. William G. Spiller at the hospital of the University of Pennsylvania. A reliable history cannot be obtained because of the patient's retarded mental development and the questionable accuracy of the observations of his parents. There was grave retardation of mental development, with inability to walk, talk, dress himself or control the urine and feces until the age of puberty or later. The parents say that the symptoms were first noticed at the age of 10 months, when the patient had an acute illness of unknown nature accompanied by high fever. At the age of 23 he began to have tonic convulsions, which would occur spontaneously or as the result of anything startling to him. The attacks occurred as often as two or three times daily; sometimes they occurred during the night.

Examination.—The clinical picture is that of residual infantile cerebral diplegia. The patient's mentality is that of a low-grade imbecile; he exhibits spastic diplegia of a mild grade, accompanied by a slight tendency to athetoid movements of the left hand on attempted voluntary movement of that member and in association with voluntary movements of the other limbs. The tendon reflexes are exaggerated; there is a sight degree of motor weakness; speech is unintelligible. The factors that have been observed to precipitate a fit include flicking the ear, lightly slapping the face and producing an unexpected sharp noise, such as that caused by dropping a spoon on a china plate. That noises do not invariably cause an attack, however, is illustrated by the fact that, during the course of a demonstration before a class of students, an attempt to induce a seizure by smashing a heavy glass plate directly behind the patient met with complete failure, although the procedure caused an unexpected loud noise. Nevertheless several spontaneous

attacks and several attacks precipitated by accidentally produced sharp noises were observed during the patient's stay on the ward.

The Seizures.—The usual attack consists of an initial tonic extension and abduction of all four limbs, with drawing in of the breath, which may or may not be accompanied by an inspiratory voice sound. There is retraction of the corners of the mouth, and the assumption of a scowling and at the same time anxious expression of the face, without closure or deviation of the eyes. The arms are usually extended in front of the body; there is mild opisthotonos, and the tonic spasm becomes increasingly more powerful. After the initial inspiration breathing is suspended and the veins of the neck become distended; the face is at first flushed and then slightly cyanotic. An inconspicuous athetoid type of movement follows, consisting of pronation of the left hand, with flexion at the wrist and maintained extension at the metacarpophalangeal and phalangeal joints. The left arm becomes adducted and flexed at the elbow, and finally the fingers become tonically flexed. There may be similar but less distinct athetoid movements of the right arm. An expiratory phonation which sounds like a half-sigh and half-laugh then occurs and marks the conclusion of the attack. Muscular relaxation and a forced sighing laugh follow.

During the seizure there is a fairly profuse flow of saliva; the pupils become dilated and do not react to light; it is probable that consciousness is distributed to some degree. Most of the attacks last for from fifteen to forty-five seconds. No incontinence of urine has been observed. Following an attack there is no distinct change in the voluntary motor power of the limbs as compared with the ordinary power; there is no demonstrable change in the mental condition immediately following a seizure. (On the light flicking of the pinna of the patient's ear without forewarning him, a typical seizure occurred and lasted about twenty-five seconds.)

The attacks are regarded as epileptic; it is probable that they are symptomatic of the same pathologic condition of the brain as is indicated by the signs of cerebral diplegia, i.e., multiple areas of scarring. The case is presented as of interest principally because of the effect of psychic factors such as surprise and fright in precipitating seizures.

DISCUSSION

DR. WILLIAM B. CADWALADER: This patient was admitted to the University Hospital last winter, was carefully studied at that time and was readmitted recently for further study. During the last year there has been no change in the man's condition. Dr. Cooper has reported that the patient's parents insist that his birth was normal and that he developed normally during infancy until the tenth month, when the abnormalities were noticed. The patient's parents are ignorant, and I do not think that their statement is reliable. I, as well as others, regarded the case as one of congenital lack of cerebral and mental development, with epileptic seizures. The epileptic attacks are always tonic and are peculiar in that they can be brought on by a sudden noise or by unexpected stimuli, such as striking or slapping the back or head, as has been demonstrated by Dr. Cooper. I saw the patient of Dr. Gordon Holmes in London when he was presented before the joint meeting of the British and American Neurological Associations in August, 1927. The case was recorded as probably one of epilepsy. It is well known that a sudden noise or sudden stimuli associated with the element of surprise may sometimes provoke epileptic seizures. This is also true of hysterical fits. It is interesting to recall that Foerster observed that sudden noises sometimes provoke epileptic attacks in cases in which there has been an injury of the left cerebral hemisphere, particularly in the region which he distinguished as number 22, that is, the area occupied by the angular gyrus.

DR. ALFRED GORDON: Did the patient ever injure himself during an attack? The position of the body during the demonstrated seizure resembled that in extrapyramidal lesions.

DR. WILLIAM G. SPILLER: The convulsions in this patient are tonic and never clonic and are of the type which I designated in my paper in *Brain* (**50**:171, 1927) as subcortical epilepsy.

DR. COOPER: The attacks apparently have been tonic, never clonic, since the onset of the seizures at the age of 23 years. The seizures have been invariably like the one just seen; they have occurred spontaneously, sometimes during sleep and sometimes as the result of noise or surprise. The rather severe abrasion of the skin on the right wrist and the scar on the left wrist are the results of injuries during attacks. Undoubtedly the patient has multiple lesions which include the extrapyramidal system, as is suggested by the mild diplegia, the posture and the moderately athetoid type of movements that occur on voluntary innervation.

I believe that the seizures are tonic epileptic attacks resulting from multiple lesions of the brain. The possibility of an epileptogenic skin zone could not be confirmed by the usual examinations. Rather intense painful stimuli were applied to the skin of the ear, scalp, neck and face particularly, as well as to that of other parts of the body, in testing the condition of the vital sensations. Unusually strong stimulation was applied because of the difficulty of holding the patient's attention otherwise. Such stimulation presented no fit. I am inclined to think that it is necessary to startle the patient, rather than merely to irritate any particular skin zone, in order to induce an attack.

MULTIPLE CEREBRAL CALCIFICATIONS. DR. F. W. SINKLER and DR. S. J. SPERLING.

F. R., a white boy, aged 18, was admitted to the Philadelphia General Hospital, in the service of Dr. F. W. Sinkler, on Jan. 5, 1933, because of tantrums. The history, obtained from his mother, is as follows: He was born at full term, with the cord wrapped twice about his neck, and was extremely cyanotic. The attending physician scarcely succeeded in reviving him by artificial respiration. Although physical development was apparently normal, except for the smallness of his head, mental development was always retarded. He did not talk or acquire proper habits of elimination until the age of 5 years. He began to walk at 20 months of age. At 10 years he entered public school, but was after three months transferred to a special school for the mentally retarded; he continued there until he was 14 years of age.

A slight twitching of the eyelids was noted at the age of 5 years; a physician said that he had astigmatism. He has always had a slight ptosis of the upper eyelids. The left eye has turned outward for the past few years, and he has turned his head to the right when looking at objects. About April, 1932, it was observed that he ran into objects and that his vision was poor; this condition has progressed so that he is now totally blind. During the past few months the eyes have been noticeably inflamed. He has been good natured and obedient until the past few months, since when he has had tantrums lasting a few minutes and characterized by shouting, cursing, pugnacity and throwing objects about. Several of these attacks have occurred on the wards; he attributes them to the fact that he is confined too much. He had measles, chickenpox, mumps and pertussis in childhood; in none of these were there symptoms suggestive of encephalitis. According to the family physician, the father had a syphilitic infection at the time of marriage, and the mother had alopecia suggestive of a syphilitic infection. There is some question whether the boy had positive serologic reactions. When he was 8 years of age his mother, on her own initiative, gave him inunctions which were presumably mercurial, for about three months.

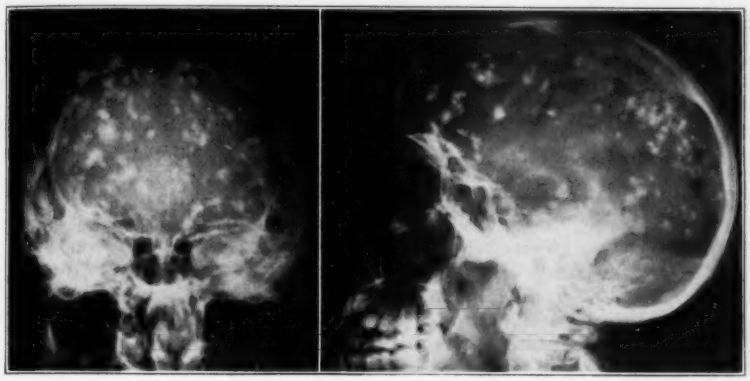
The parents are well; a sister, aged 16, and a brother, aged 14, are well and are said to be of normal mentality. A brother was accidentally killed in childhood. There is no history of mental or nervous disease in the family.

Physical examination gave essentially negative results except for a small skull and bilateral keratoconjunctivitis with linear ulceration of the corneas. The pupils, 2 mm. in diameter, were fixed to light. Cataractous deposits were found in the right eye. There is doubt whether the inflammation is of syphilitic origin.

Neurologic examination gave essentially normal results, although the patient cooperated poorly. Facial, lingual and palatal conditions were normal, as were hearing and smell. All the deep tendon reflexes were active and equal, and there was absence of ankle clonus and of Babinski or Hoffmann signs. The abdominal reflexes were present. There was no atrophy or weakness of the limbs, and there was no dysmetria. The sensations of touch and pain were apparently normal, and there was no aphasia or dysarthria.

Psychometric examination revealed a mental age of 5 years and 9 months, with an intelligence quotient of 35.9, which places the patient in the group of middle grade imbeciles. He was able to count correctly only up to 12 and was unable to reverse two digits or recall the number of fingers on his hands. He was, however, able to say simple words, to discriminate between right and left and between morning and afternoon.

Urinalysis gave essentially negative results. The blood sugar content was 118 mg.; the urea content was 15 mg.; the calcium content was 10.2 mg., and the phosphorus content was 4.4 mg. The Kahn test of the blood and the Wassermann test of the spinal fluid both gave negative results. The spinal fluid showed no cells, but there was a trace of globulin.



Roentgenograms of skull showing shadows of calcifications scattered throughout the brain.

The striking feature of this case is seen in the roentgenograms of the skull. There are numerous, irregular, circumscribed, calcified shadows scattered throughout the brain. The skull is much smaller than normal. The sella turcica, clinoids and dorsum sellae are normal, there being no evidence of increased intracranial pressure or of calcification of the suture lines. Roentgenograms of the chest and pelvis are normal.

The x-ray films have been seen by many roentgenologists in this city and elsewhere, including Drs. Chamberlain, Pancoast, Pendergrass, Pfahler, Sosman, Edeiken, Holmes and Ostrum. These observers suggested as diagnostic possibilities multiple calcified tuberculomas, calcified parasites and cerebral calcifying endarteritis. The multiplicity of the lesions, their presumably long duration, the absence of tuberculosis elsewhere and the excellent general physical condition of the patient render tuberculomas extremely unlikely. Parasitic lesions, particularly those of cysticerci, are much smaller, are more spherical and are often present elsewhere in the body. The type of cerebral calcification epilepsy (endarteritis calcificans cerebri) reported by Geyelin and Penfield (ARCH. NEUROL. & PSYCHIAT. **21**:1020 [May] 1929) in an interesting family group bears a resemblance (particularly their case 4) to this case, but closer study shows that the calcification which they observed was typically vascular, whereas in the present case many roentgenologists

state that the calcification is not vascular. Franke-Stehmann's (*Arch. f. Psychiat.* **96**:634, 1932) second case resembles this one somewhat, but the similarity is only partial. Dr. Pancoast pointed out in this case that on stereoscopic examination the calcifications appear to be subcortical and in a location similar to that in case 3 of Geyelin and Penfield, in which the calcification was observed to be between the white and gray matter. Cases showing calcification of the smaller cerebral vessels and capillaries, with relatively normal larger vessels, generally seen in younger persons who are epileptic, have been reported by Bamberger (cited by Rokitansky in Lehrbuch der pathologischen Anatomie, Vienna, W. Braumüller, 1856, vol. 2, p. 472), Mallory (*J. Path. & Bact.* **3**:110, 1896), Hansemann (*Verhandl. d. deutsch. path. Gesellsch.*, 1899, p. 399) and Bassoe and Hassin (ARCH. NEUROL. & PSYCHIAT. **6**:359 [Oct.] 1921). A fourth diagnostic possibility is multiple calcified cerebral hemorrhages, possibly related to birth trauma.

MULTIPLE CRANIAL NERVE PALSIES. DR. WALTER FREEMAN.

Numerous combinations of cranial nerve palsies have been described, and a baffling array of names has been attached to them. More important than the possibilities of combinations is the localizing value of the various syndromes. The problem is relatively simple when several adjacent nerves are paralyzed and when the disorder is limited to one side. For exact localization the question of intracranial versus extracranial disorder is of less importance than that of intracerebral versus extracerebral disorder, since growths within the brain stem are surgically unapproachable, while extramedullary growths may be attacked. This was forcibly demonstrated to me several years ago by the case of a woman who had paralysis of the fifth, sixth, seventh and eighth cranial nerves. An operation for the removal of a suspected acoustic tumor was scheduled, when Dr. Spiller noted paralysis of the lateral associated movements of the eyes, with preservation of the convergence reaction. This constituted the syndrome of Foville, by which it was manifest that the median longitudinal fasciculus was affected. The operation was canceled. Necropsy showed a glioma of the pons.

While the localization of the disease is usually fairly simple, the nature is not always evident. The differential diagnosis rests on collateral findings. For instance, a meningitic disorder will reveal itself in the spinal fluid, a traumatic lesion is usually differentiated by the history, and the finding of a pharyngeal growth indicates trouble about the base of the skull. The x-rays cannot be relied on greatly on account of the difficulty of securing satisfactory visualization of the base of the skull, but they may yield important information. Diagonal pictures of the skull and postero-anterior views throwing the profile of the petrous ridges above the orbits are the most satisfactory.

Report of Case.—Mary S., a white woman, aged 69, was admitted to St. Elizabeth's Hospital in Washington, in 1919, about two years after the onset of a psychosis characterized by delusions of sin and of people spitting at her; she was depressed, prayed constantly for forgiveness and was seclusive and at times resistive. Her father and a sister had died of cancer. Physical examination revealed nothing abnormal except emaciation, which was increased by her refusal to eat. At one time she weighed only 76 pounds (34.5 Kg.); she had to be fed by means of a tube for considerable periods.

The first note of physical disorder was made by Dr. Billard on June 2, 1932, when he reported paralysis of the right external rectus and partial paralysis of the upper lid, with a normal fundus. About a month later it was noted that her right eyelid was kept spasmodically closed. On account of lack of cooperation it was difficult to examine her, but on October 18 a report was made.

The patient was inattentive and resisted the examination of certain parts. She said that she could not see out of the right eye; the lid had been drooped since May; when it was raised she saw double. The sense of smell was preserved on the right; the fundus showed a small degree of arteriosclerosis and a slight irregularity in the outline of the disk, but the color was normal throughout. The right eye was in midposition; there was no movement in any direction; the pupil

was large and inactive. The patient resisted opening of the mouth. The contraction of the masseter muscle seemed less on the right than on the left. The face was insensitive to pinprick on the right but not on the left, and deep pressure was felt only to a moderate degree; the cornea was insensitive. The right side of the face was completely paralyzed, and the mouth was drawn to the left when it was opened or when the patient smiled. The right eyelid could be opened easily against the patient's will. The sense of taste could not be tested owing to lack of cooperation. The right auditory canal was reduced to a narrow vertical slit, and the tympanic membrane could not be seen. The right ear was completely deaf; there was lateralization of sound to the left. The palate was elevated on the left side; there was a finger-like projection of irregular, somewhat granular character behind the right side, which was firm but which bled easily. This was obviously a new growth. Pharyngeal and palatal reflexes were absent on the right, although they were present on the left. Phonation was fairly adequate, and the vocal cords behaved normally. The sternomastoid muscles appeared to be about equally well developed on the two sides. There was a firm mass beneath the upper end of the muscle on the right side, presumably a metastasis. The patient would not protrude her tongue adequately, but no paralysis or atrophy was noted, and articulation was fairly good considering the lack of teeth.

The upper limbs were wasted; the little finger of the right hand and the ring finger of the left hand showed beginning Dupuytren's contraction. The reflexes were increased about equally in the upper limbs, but no special signs were elicited. Sensibility to pinprick, vibration and movement was preserved in the fingers, and coordination was good, although tremors were present. The lower limbs were thin, and the reflexes were increased. There was an abortive ankle clonus on the left. The Babinski sign was negative. Pinprick, vibration and movement were felt in the toes, and the patient stood well, even with the eyes closed.

During the past few months there has been progressive enlargement of the pharyngeal growth, occlusion of the posterior nares and weakening of the sternomastoid muscles on the right. At one time acute parotitis developed on both sides; this subsided without suppuration. Swallowing has become increasingly difficult; the metastatic nodes have increased in size, and the conjunctiva is markedly inflamed.

Comment.—The disproportion between the extensive paralysis of the cranial nerves and the slight indications of disturbance in the brain is indicative of a disorder that is primarily extracranial. In such a condition, with involvement of many nerves, it is striking that no complaint of pain has been made. This is explainable only on the basis of the patient's mental condition, since growths in this location are notoriously painful. There is no question of extirpating the growth; death will come eventually through aspiration pneumonia when the swallowing mechanism is put out of commission. The development of a tumor in the nasopharynx in a patient subjected to frequent feedings by means of a tube is of interest from the standpoint of possible traumatism; equally interesting is the "wisdom of the body," to use Cannon's phrase, in causing a tumor to grow in this location in a person who refused to eat normally.

DISCUSSION

DR. JOSEPH C. YASKIN: A year ago I presented the case of a young woman with bilateral involvement of the fifth cranial nerve. In this case roentgenologic studies revealed a tumor in both middle fossas, extending into the anterior fossa. There was progressive involvement of the first, third, fourth, fifth, sixth, seventh and eighth cranial nerves on the left side and of the third, fourth, fifth and sixth cranial nerves on the right side. After intensive treatment by irradiation this patient made a temporary recovery. Several months later, however, the symptoms recurred, and the tumor protruded through the left nostril.

DR. FREEMAN: This patient has never complained of pain. We cannot see any reason for it other than the psychosis. The growth should cause severe pain.

UNILATERAL CHOKED DISK DUE TO CHRONIC TONSILLAR INFECTION. DR. GEORGE WILSON and DR. W. F. DARKES.

Choking of the disks (papilledema) is in the majority of cases (probably 90 per cent) due to cerebral neoplasm. Unilateral choking may occur with a tumor of the brain, although as a rule involvement of the other disk soon occurs. We have observed two recent cases of tumor at the base of the right frontal lobe with unilateral choking of the right disk of 5 and 4 diopters respectively: the left disk in both instances was normal. When one is confronted with a patient with unilateral or bilateral choking of the disk, the initial and most important step is to rule out tumor of the brain; after that has been done search must be made for the more uncommon causes, as, for example, acute and chronic infections, toxemias, certain diseases of unknown etiology (such as multiple sclerosis) and syphilis. The concern in this presentation is with unilateral choked disk due to chronc infection in the tonsils, of which we have seen two instances.

Hansell (*M. News Phila.* **49**:144, 1886) reported two cases of acute choked disk with sudden loss of vision and a rapid return to normal with treatment; he thought that the cause was a rheumatic infection. In one of these cases the choking was unilateral. The papilledema was explained by the exudation of serum into the fibrous coat of the nerve, which produced sufficient local pressure to cause swelling. De Schweinitz (*Tr. Philadelphia County M. Soc.* **60**:247, 1886; *Tr. Philadelphia Polyclin.* **5**:491, 1896) reported a case of acute unilateral choked disk which at that time he thought was due to exposure. He reviewed the case in 1896; he then believed the disorder to be due to a rheumatic infection. Suker (*Ophth. Rec. Chicago* **26**:278, 1917) cited a case of unilateral optic neuritis as the result of chronic infection in the tonsils. The case was that of a young woman who, in March, 1917, noticed blurring of vision in the left eye, which resulted in loss of vision in that eye in the course of a few days. The left disk was markedly swollen, although Suker did not state the exact amount of swelling. Seventy-two hours after the tonsils were removed vision in the affected eye was normal; in four weeks the swelling in the disk had disappeared.

Ball (*Arch. Ophth.* **49**:335, 1920), Carpenter (*Am. J. Ophth.* **1**:787, 1918) and Harry (*Brit. J. Ophth.* **6**:216, 1922) have reported similar cases, although in some the optic nerves were involved bilaterally. Harry stated that the characteristic features of choked disk due to chronic tonsillitis are acuteness, intensity, the unilateral character and the completeness with which the condition clears up after the removal of the tonsils. In Carpenter's case, reported in 1918, blindness in one eye disappeared after the removal of the tonsil on the same side. It is well known that a chronic infection can produce involvement of a single joint, and it is therefore not unreasonable to assume that a focal infection might implicate one optic nerve. The investigations of Brown and Irons (*Tr. Am. Ophth. Soc.*, 1918) and of Rosenow (*J. Infect. Dis.* **17**:403, 1915) show the frequency with which iritis and other ocular infections may be caused by streptococci and other organisms. We are fully aware that it is possible for any chronic infection to be the cause of optic neuritis, but the two conditions that are reported here are clearly due to infection in the tonsils.

CASE 1.—Mrs. M., a white woman, aged 52, was referred for treatment by Dr. Spangler and Dr. Chapin Carpenter on March 19, 1931. Her medical history and family history are unimportant, except for the fact that she had suffered from repeated attacks of tonsillitis and for the past few years has had mild arthritis in both hands. On March 12, 1931, she noticed, while reading the morning paper, that vision in the left eye was poor. She had no other symptoms, such as headache, vomiting or double vision.

General neurologic examinations gave negative results except for badly diseased tonsils, signs of mild chronic arthritis in the joints of the hands, a systolic murmur at the apex of the heart and pain when the left frontal region was percussed. Dr. Carpenter examined her eyes on March 12 and reported that there were no external abnormalities; the ocular movements were full; tension and pupillary reactions were normal; vision, corrected, was 5/5 for the right eye and 5/12 for

the left eye. Ophthalmoscopic investigation of the right eye showed the media to be clear and the nerve normal; on the left side the media were clear and the nerve presented a choking of 2 diopters. The vessels were congested and were buried at the margin of the disk, but there were no hemorrhages. Two days later hemorrhages appeared on the disk. The right eye remained free from involvement. The visual fields showed a central scotoma for colors and for white. A week later Dr. Carpenter reported fresh hemorrhages and an increase of the swelling to 4 diopters.

A roentgenogram of the skull taken by Dr. George Pillmore showed no evidence of sinus disease or of increased pressure. The size and shape of the sella were normal. The blood count, the blood chemistry and the urine were normal. The reaction of the blood to the Wassermann test was negative. The spinal fluid was normal except that the total protein content was increased to 3 units (the normal content being 1.5 units).

In the absence of signs indicative of tumor of the brain, sinus disease, syphilis and any general toxic factor such as might occur in renal disease, it was decided to remove the diseased tonsils. This was done by Dr. George Coates on April 1. Vision began to improve almost immediately. Dr. Carpenter reexamined the patient on April 15 and reported that the choking had subsided to 3 diopters. No fresh hemorrhages had occurred. The exudate had been absorbed, and the outline of the disk was almost visible in the upper nasal portion. Vision was corrected to 5/9, and small test type could be read, though with a slight central blurring. On July 9 Dr. Carpenter reported that vision of the left eye, corrected was 20/15. The visual field in the left eye was almost normal; there was only a slight central scotoma. The hemorrhages had been completely absorbed, but some atrophic changes in the choroid remained. About 0.5 diopter of swelling remained.

CASE 2.—Mrs. J., a white woman, aged 28, was referred for treatment by Dr. H. G. von Goldberg on Oct. 19, 1931. Her history has no bearing on the present illness, although she had had tonsillitis on one occasion. Four weeks before examination she was perfectly well. She awoke one morning with pain in and over the right eye; vision of the eye was blurred. The pain lasted for two days. At the time she was seen she complained of pain when she moved the right eye. She had seen double on one or two occasions.

General and neurologic examination showed nothing significant, with the exception of enlarged and diseased tonsils. Dr. von Goldberg reported that when he first saw the patient, on October 10, vision in the right eye was 6/12, and in the left, 6/6. There was 4 diopters of choking in the right eye; the veins were engorged, and there were small areas of blood dotting the head of the nerve. The left eye was normal; there was no ocular palsy. On October 17 the swelling of the head of the nerve had increased to 6 diopters, and vision had dropped to three lines.

The patient was referred to the University Hospital. Dr. Karl Houser could find no evidence of sinus disease, and Dr. Pancoast's roentgenogram of the sinuses showed normal conditions. A roentgenogram of the skull showed no increased pressure, and the pituitary fossa showed no deformity or enlargement. The urine, blood count and blood chemistry were normal. The reaction of the blood to the Wassermann test was negative. The spinal fluid was normal except that the total protein was 2.5 units. In view of the experience in case 1 and in view of the negative signs for syphilis, a sinus disease or tumor of the brain, a tonsillectomy was performed by Dr. Karl Houser on October 23. A week later the patient reported definite improvement in vision. Dr. von Goldberg noted on October 31 that vision in the right eye had improved to 6/8 and that the swelling in the head of the nerve had subsided to 3 diopters, with corresponding improvement in the surrounding area. On November 7, two weeks after removal of the tonsils and seven weeks after the onset of the trouble, vision in the right eye was 6/6; the margins of the nerve were clearly defined, except for a small area above and to the nasal side; the hemorrhages on the head of the nerve were absorbed, and the vascular and other pathologic conditions had disappeared. When last heard from on Jan. 21, 1933, the patient reported that her vision had remained normal.

Comment.—Few bodily complaints cause as much concern to the patient as disturbance of vision. Sudden loss of vision in one eye is alarming, and when optic neuritis or choked disk is discovered the responsibility of the physician is great. The triad of symptoms—headache, vomiting and choked disk—is common in tumor of the brain. Choking of the disk is usually bilateral, and, while an attack of sudden temporary blindness may and does occur, the loss of vision as a rule is gradual. Furthermore, there is a possibility of perfect vision with a high degree of choking of the disk when the choking is due to a tumor. The characteristic features in the cases studied by us and in those reported by others are sudden loss of vision, rapid increase in the swelling and unilateral involvement, although bilateral involvement has been reported. Removal of the tonsils in both cases reported here resulted in prompt improvement and a return in a relatively short time to a normal condition. No time should be lost in arriving at a diagnosis. If the swelling of one or both optic nerves is the only physical sign present, and if the patient does not have headache or other symptoms suggestive of tumor of the brain, there should be no haste to take an encephalogram or a ventriculogram or to perform a decompression operation. Cases of the kind reported have probably helped to swell the number of those referred to naïvely by the neurosurgeons as "pseudotumor."

NEW YORK NEUROLOGICAL SOCIETY

Regular Meeting, Feb. 7, 1933

HENRY ALSOP RILEY, M.D., *President, in the Chair*

THE DEATH INSTINCT IN SOMATIC AND PSYCHOPATHOLOGY. DR. SMITH ELY JELLIFFE.

In my early thirties, while still at the beginning of my medical career and doing work outside of the actual practice of medicine, I became editor of the *Medical News,* one of the oldest of the medical weeklies of the United States. The annual meetings of the American Medical Association were events of much moment to the editors of the four or five medical weeklies then existing, and hence I visited them. In 1905, the annual meeting took place at Portland, Ore., and it was then that an occasion was offered to visit Alaska; I traveled as far north as the Great Divide, down which the gold rush descended in the memorable push to the sands of the Yukon.

One of the impressions of the trip, which constitutes the beginning of this contribution, was the vivid picture of the mad climb of the salmon up the rivers of the western coast carrying out their strange and fatal journey during which the instinctive impulses of life and death fought out their implicit patterns in grotesque antithesis. To one interested in natural history it was an old story, but not until the billions of salmon were seen caught in the grip of the death instinct after their frantic struggle to procreate did the whole adventure possess any vivid reality. Nor did it seem to have any intimate relation with human life until the stimulating thoughts of Freud gave new meaning to many of the problems of medicine. When, almost twenty years later, I read Freud's "Beyond the Pleasure Principle" (1920) and, later, his "Ego and the Id" (1923), the whole series of conceptions connected with the strange behavior of the salmon was recalled.

It has been a commonplace experience to note a frank psychotic expression of conflict clear up and find as an alternative in the same patient a disturbed renal function, a disorder of the skin, diabetes, a diseased sinus or a hyperthyroid state. In a number of cyclothymic persons there was observed a regular replacement of the one group of manifestations by the other. When the neurotic or psychotic phenomena were in the ascendant the anomalous somatic behavior sank beneath

the horizon, to emerge in definite form as the more evident mental expressions in turn faded out of the picture. And thus the cycle of replacement went on.

Finally the generalization became clearer as the concept of the holistic nature (Smuts, J.: Holism and Evolution, New York, The Macmillan Company, 1926) of man became more evident and the human being as a whole was seen to be in a constant state of unstable equilibrium. Functional variability as an evidence of the struggle of systems of energy within and without the body was the rule rather than the exception.

Medical science in the past century has opened up a clearer vision of those systems of energy from without (trauma—infection) and from within (metabolic), but it was not until the genius of Freud developed a similar unitary understanding of the dynamics (economics) of the mental systems from without, as bound up in customs, folklore, religions, caste, class, etc., and those from within (identification with parents—Oedipus structural patterns, instinct drives, etc.) that a true holistic view of many disease processes could be gained. Then came a long period of close and intense analytic work with partial and localized forms of instinctual disintegration in so-called organic disease.

Above all it might be recalled that the human machine is no simple jack-in-the-box. As it takes great patience persistently to dissect the mixed and subtle psychic processes, infinitely more complex than any other in human activities, so it would require volumes of the words and concepts now available to report such micropsychic dissections. It is revealing that Freud's "Fragment of an Analysis of a Case of Hysteria," as he called it, fills 146 large octavo pages in English translation; for me there is not a useless phrase in the whole presentation.

The point to be emphasized in regard to personal studies is that the sadistic (hostility) component (death instinct) has operated like a castration or partial death of an organ or of certain functions of an organ and has thus brought about disharmony of function. In some instances it has been more malignant (irreversible) than in others. I have subsumed such cases as instances of malignant conversion (irreversible processes) in which the conversion mechanisms follow in part those seen in the less malignant hysterias (reversible processes). The quantitative factors which have interfered with an adequate compromise of the conflict can be evaluated chiefly through the conception of the tyranny of the mechanism of the superego. The law of retribution (*lex talionis*) has been too rigorously carried out. Just how this comes to be, chiefly because the libido regression becomes fixated in the early organic erotic stages and hence involves the adjuvant instinctual frustrations, cannot be further elaborated here. It is not my intention at this time to attempt to generalize on these various observations in different medical fields or to follow along the same pathways with newer ones. In the present communication the method is reversed, and an attempt is made to present an idea looking toward an understanding of how certain procedures which are in widespread use in the treatment of certain mental disorders can be thought of as accomplishing some of their beneficial results.

As one traces instinctual processes to their origins one inevitably discovers the work of the sexual impulse, the work of Eros, as Freud has aptly termed it. "The clamor of life proceeds for the most part from Eros." Sadistic or destructive components (death instincts—thanatos) are always fused with Eros but are usually "mute." In the melancholias they "speak," and also, quite volubly, in the so-called paranoias and compulsion neuroses, in which last type they show the destructive tendencies behind all fanatic ceremonial. Such fixed ceremonials are exemplified in history in stories of the sense of guilt and the need for punishment, from the time of the account of the expulsion from the garden of Eden to the present. The voice of thanatos, i. e., sadism, which operates chiefly though not exclusively through the repressions of the superego (conscience, in part), does not often break through into the conscious ego system as the pain of depression or phobias, as compulsions, as conversions or as projections in hallucinatory or delusional formations—all of which have come to be evolved economically as saviors of the organs of the body.

When a complete release of all the sexual tensions takes place, as is seen in the bit of biologic behavior which has been mentioned as starting this inquiry, namely, the death of the salmon after the discharge of its sexual substances, the disintegration of instincts becomes most notable, and death takes place.

Freud states on this point: "The Id, guided by the pleasure principle, that is by the perception of 'pain,' guards itself against these (libido) tensions in various ways. It does so in the first place by complying as swiftly as possible with the demands of the non-desexualized libido; i. e., by striking for the gratification of the directly sexual trends. But it does so further, and in a far more comprehensive fashion, in relation to one particular form of gratification which subsumes all component claims—that is, by discharge of the sexual substances, which are saturated conductors, so to speak, of the erotic tensions.

"The ejection of sexual substances in the sexual act corresponds in a certain degree with the separation of soma and germ plasm. This accounts for the likeness between dying and the condition that follows complete sexual satisfaction, and the fact that death coincides with the act of copulation in some of the lower animals. These creatures die in the act of reproduction because, after Eros has been eliminated through the process of gratification, the Death Instinct has a free hand for accomplishing its purposes. Finally, as we have seen, the Ego, by sublimating some of the libido for itself and its purposes, assists the Id in its work of mastering the tensions.

"There are certain people who behave in a quite peculiar fashion during the work of analysis. When one speaks hopefully to them or expresses satisfaction with the progress of the treatment, they show signs of discontent and their condition invariably becomes worse. One begins by regarding this as defiance and as an attempt to prove their superiority to the physician, but later one comes to take a deeper and truer view. One becomes convinced, not only that such people cannot endure any praise or appreciation, but that they react inversely to the progress of the treatment. Every partial solution that ought to result, and in other people does result, in an improvement or a temporary suspension of symptoms, produces in them for the time being an exacerbation of their illness; they get worse during the treatment instead of getting better. They exhibit the so-called negative therapeutic reaction.

"There is no doubt that there is something in these people that sets itself against their recovery and dreads its approach as though it were a danger. We are accustomed to say that the need for illness has got the upper hand in them over the desire for health. If we analyze this resistance in the usual way—then, even after we have subtracted from it the defiant attitude toward the physician and the fixation on the various kinds of advantage which the patient derives from the illness, the greater part of it is still left over and this reveals itself as the most powerful of all obstacles to recovery, more powerful even than such familiar ones as narcissistic inaccessibility, the assumption of a negative attitude toward the physician or a clinging to the advantages of the illness.

"In the end we come to see that we are dealing with what may be called a 'moral' factor, a sense of guilt, which is finding atonement in the illness and is refusing to give up the penalty of suffering. We are justified in regarding this rather disheartening explanation as conclusive. But as far as the patient is concerned this sense of guilt is dumb; it does not tell him he is guilty; he does not feel guilty; he simply feels ill. This sense of guilt expresses itself only as a resistance to recovery which it is extremely difficult to overcome. It is also particularly difficult to convince the patient that this motive lies behind his continuing to be ill; he holds fast to the more obvious explanation that treatment by analysis is not the right remedy for his case."

The issue that presents itself in the present suggestion is the direct corollary of all this and in a sense an ambivalent one. If one can hurt, that is castrate, the organ which is being used as a scapegoat, in whole, or, preferably, in part, the punishment may be made to fit the crime, theoretically at least.

One is faced, however, with some extremely interesting types of recovery from severe mental illnesses following acute somatic illness. Following an attack of typhoid fever, influenza or pneumonia, an accident, a severe operation or blood letting, an extreme purgation, and sometimes even such minor events as tonsillectomy, the pulling of teeth, thyroidectomy or appendectomy, certain mental illnesses, some of long standing, have often cleared up. The literature is vast and need not be summarized here. The relation of the disease process to the economics of the displacement of the libido and to instinctual fusion or defusion is not exactly statable, even though the principle is obvious.

In certain claimed results following colonic resection in schizophrenia, for example, one can follow the issues here outlined to advantage. Undoubtedly, certain favorable results have taken place. They are not, in the opinion of the reasoning here sketched, the results of modifying focal infection. The beneficial changes have resulted from carrying out the castration threat on a particular organ which has been made the specific medium for sadistic encystment of the primary narcissistic libido, namely, the lower bowel. From the earliest days of the child's training the pleasure zone of the hostile, aggressive and sadistic components has been related to this area. The lower bowel and the motor apparatus are the chief primary organs for the expression of sadism. The symbolism of the "cities of the plain," Sodom and Gomorrah of the ancient Hebrews, points to the chief abiding place of the unsublimated, homosexual, sadistic component of man's strivings. Here is *au fond* the avenue through which the death wish can find an outlet. In mutilating this organ one punishes it; an "eye for an eye" retributory penance has been paid, and the instinctual conflict is rearranged, sometimes to advantage. Two case histories are reported illustrating the principal issues.

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FREUD'S MORE RECENT CONTRIBUTIONS TO PSYCHOANALYSIS. DR. A. A. BRILL.

Freud's metapsychologic writings, which appeared in a series of papers between 1914 and 1917, were given out as an elucidation and clarification of the deeper processes of psychoanalysis. Freud stated: "I propose that when we succeed in describing a psychic process, in its dynamic, topographic and economic relationship, we should designate it as a metapsychologic presentation." All these mechanisms were clearly demonstrated in simpler form in Breuer's and Freud's first contributions on hysteria. Freud's new series of lectures are the chapters "Revision of the Dream Theory," "Dreams and Occultism," "The Analysis of the Psychic Personality," "Anxiety and the Instinctive Life" and "Femininity." In the analysis of the psychic personality Freud traces the development of the superego, the highest mental evolution of man. Its abnormal activity is readily seen in delusions of observation, as manifested in paranoid conditions. But the same self-observation is regularly seen in the normal structure of the ego; the average person, too, regularly watches and criticizes himself, especially when he commits a wrong. This force is nothing but conscience. The force of the superego is also manifested in other psychotic conditions, notably in melancholia, in which there is a definite struggle between the ego and the superego. Here the ego confesses its weakness and unworthiness and yields to the punishment of the superego, a situation which often results in suicide. The familiar moral feeling of conscience, according to Freud, represents a feeling of tension between the ego and the superego. In contrast to sex, which appears in the beginning of life, conscience or morality is nonexistent in childhood. Moreover, morality seems to be a periodic or transient phenomenon. The child is not moral but gradually acquires this feeling through the influence of its parents, who govern the child by means of love and threats of punishment. Real anxiety is thus the precursor of conscience-anxiety. The superego is the legitimate heir of parental force and contains, therefore, the idea of punishment and strictness. The transformation of the parental influence into the superego is based on the process of identification. The boy identifies himself with his father by assimilating into his ego the strange or foreign ego of the father. The creation of a superior force in the ego is also intimately connected with the fate of the Oedipus complex; the superego is thus the heir of the most

important emotional attachments of childhood. The superego is the representative of all moral restrictions, the advocate of all strivings for perfection.

The only revision of Freud's theories in thirty-three years concerns the theory of wish fulfilment, which has always been questioned by some investigators. Freud replies by dividing dreams into wish, anxiety and punishment dreams. It is in the last two types of dreams that the theory of wish fulfilment is disputed. In the punishment dream the wish fulfilment does not emanate from the instinctive feelings, as is usually the case, but from the critical and censorious force of the superego. There are, however, two types in which the function of wish fulfilment is either imperfectly realized or fails altogether. The first type includes dreams which reproduce infantile scenes of painful, anxious and disappointing impressions, but these infantile experiences contain all the unfulfilled wishes which furnish the motive power for the formation of dreams throughout life. In their effort to break through they drag along the painful material which was connected with early childhood. In traumatic neuroses the wish tendency is absent; painful traumas actually experienced recur frequently in dreams. Freud frankly states that here the function of the dream fails, and he counsels those who wish to take note of this exception to modify his statement that "the dream is an effort at wish fulfilment."

Concerning dreams and occultism, Freud states that after painstaking investigation of occult phenomena he wishes to assume a broad-minded attitude toward such problems. The cases he cites were not fully convincing to himself, nor are they to the reader. After giving examples of telepathy, fortune telling, etc., he states that it would seem that some fortune tellers read, as it were, the unconscious wishes of their clients.

A Neurosis À Deux. Dr. Clarence P. Oberndorf.

A case of induced neurosis in a couple who had been married eight years was presented. The main symptom of the neurosis in each person consisted of a sensation of whirling. They also indulged in an unusual mutual sexual perversion. The cases were presented as contributions to the question of blood relationship and previous psychopathy in cases of folie à deux and more particularly in relation to the influence of identification in the assumption of symptoms by the persons concerned in induced psychopathy. The circumstances in this instance precluded blood relationship of the two persons; the social background and even the religions of the husband and wife were different. On the other hand, the history of each revealed marked neurotic traits from childhood, especially in the husband, who had been afflicted with the perversion of immersing himself in a bathtub attired in his clothes from the age of 8. The fact that the husband had been afflicted from boyhood and subsequently persuaded his wife to join him in the perversion would ascribe to him the rôle of inductor into the abnormal social existence which the couple led. The psychology of the inductee, although it has received little attention in literature, is as important as the more striking rôle of the inductor. In this case the basis of the folie rested on the process of identification. Psychoanalysis of the wife showed that in the folie à deux she identified herself with a lost object, namely, that of masculine striving, which had appeared early in her childhood. The husband, on the other hand, identified himself with the lost object of female identification; originally, in his bathing procedures he had identified himself with his mother. Each unconsciously found in the other the lost complementary objects. The analyses of these two persons were conducted contemporaneously, with benefit to each.

Interpretation of Dreams in the Practice of Psychoanalysis. Dr. Monroe A. Meyer.

The interpretation of dreams during the course of psychoanalytic treatment is practically useful. The value of the correct reading of dreams, both to the analyst, from the standpoint of orientation, and to the patient, from the standpoint

of insight, is illustrated by: (*a*) a series of dreams that dealt with the unconscious precipitating factor in a case of depression, (*b*) a biographic dream correlating the patient's adult psychosexual situation with infantile factors, (*c*) a transference dream that revealed the patient's unconscious attitude toward the analyst and (*d*) a dream elucidating the patient's current conflict.

DISCUSSION

DR. PAUL F. SCHILDER: The chapter in Freud's book concerning telepathy gives the impression that he is much impressed with telepathy. He says that he cannot offer definite proof of it but is inclined to believe in it. I do not agree with Freud in this respect. I am strongly of the opinion that in investigating telepathy one should not rely on impressions but should wait for at least one definite fact. I hunted for this definite fact for several years in Vienna. Many persons immediately after the war claimed to possess occult powers; I failed to find any evidence of them. Some of these persons were swindlers; others, untrained in observation, deceived themselves as well as others. I do not mean to say that telepathy is impossible; but I think that we should wait for facts. Many occurrences are rather astonishing in dreams, in the analytic hour and in daily life, but one must not forget that human beings are not very original—practically all come from the same milieu, and have the same psychic food—so that there is a uniformity in human beings which accounts for many of these phenomena. More important is Freud's statement that insects may communicate in a telepathic way with one another. There is definite experimental material in regard to the language of bees. The excellent investigations of von Frisch are revealing to every biologist and psychologist. When bees have found flowers which give a great deal of honey they fly back and start to dance. Thus they communicate to others by movements of their bodies, which are transmitted to the bodies of neighboring bees. These leave the beehive to search for the flowers. The communication by bodies is attained through the medium of external movements. The other bees become excited by the touch of the dancing insect. I mention these facts because one sees communication through expressive movements in the human relations of everyday life as well as in psychoanalysis. Neurotic and psychotic patients tend to feel strongly this connection between their own body images and those of others. A short time ago I observed a patient with alcoholic hallucinosis who felt that people around him passed remarks which were meant for him. When these words were pronounced he was asked to associate freely. Many sexual words, representations and thoughts came into his mind through this "forced" free association. Then his fingers started to move against his will. He believed that these movements revealed his innermost feelings and thoughts to others. There is always a close communication between body images, and the body expresses the inner tendencies of the individual. Freud has pointed out that unconscious emotions are communicated with special ease. These facts account for all the telepathic phenomena that I have observed so far. When other phenomena are observed other interpretations may be necessary. There are no reliable data of this kind yet available. Emotions express themselves in the body, and each of us is interested in his own body as well as in the body of others.

Dreams should be regarded from four points of view. First: what does the dream mean in the present history of the subject? This point has been clearly stressed in the first case of Dr. Meyer, in which the actual situation reflected itself in the dream. Though the dream is an expression of an attitude concerning the actual situation, it is more than that. It is also an expression of an infantile situation, and in his second analysis Dr. Meyer has shown clearly how the dream expresses an important part of the infantile history of the subject. But one is certainly not satisfied with these two analyses of a dream. There is a third interpretation, namely: What does the dream mean in the transference situation? What does it mean in the analytic situation? The dream of Dr. Meyer's patient, in which he is taken for the Lord, is a good example. The patient expresses all her distrust of Dr. Meyer by comparing him with the Lord in "The Green

Pastures." But even when one has considered these three possibilities there is another that is no less important, namely: What does the dream mean from the point of view of the body? This is the part of the interpretation of dreams which offers the greatest difficulties to nonpsychoanalysts. Scherner has expressed the idea that the dream means something in terms of the body. It symbolizes the mouth, breasts, urethra, anus, genitals or other parts of the dreamer or of other persons. One of the dreams reported by Dr. Meyer provides an example—the dream about the queen bee. The queen bee is not only the love object of the dreamer—the person whom he loves in an ambiguous way—but it might symbolize also her genitals. The arm of the dreamer also means another part of his body—his genitals. In a similar way the body image of the dreamer participates in every dream; the body image is the real source of symbolism in the dream. This deep-rooted symbolism provides the greatest difficulties in understanding the problem of the interpretation of dreams.

This leads me to Dr. Oberndorf's paper, in which the body image again plays many rôles. Here there is close identification between the neurotic husband and the neurotic wife. The man identifies himself with his mother, and the clothing of the mother plays an important part in this mechanism. The body image goes beyond the body; according to Head, it reaches up to the feather of the woman's hat. The body image is also the basis for identification. Human bodies take parts of the bodies of others into themselves and project parts of their own bodies into others. This occurs in every neurosis, and every neurosis is not only a neurosis à deux, but a neurosis in the community—of course in that part of the community closest to the patient, i. e., the family. Continual interchange between body images takes place. The understanding of the psychology of the body image (everyone has a three dimensional picture of himself) is also necessary for a deeper insight into the problem of conversion. Conversion does not take place in the body only. It enters into the mental image of the body—into the picture that we have of ourselves. Dr. Oberndorf's case offers interesting problems in this respect. The body has been submerged, and when one is submerged the heaviness of the body changes, especially in the legs. The body feels lighter. The feeling of lightness and heaviness of the body is also dependent on the vestibular apparatus. Later the legs become centers of auto-erotic experience. But the gravity experience which is connected with the vestibular apparatus has also been altered by events in early childhood. One must always ask what part of the body image has been emphasized—what part has been affected in early childhood. This influence may be merely psychologic or it may be more nearly physiologic. Most often it will be psychologic as well as physiologic. The part of the body image to which attention has been drawn becomes later the part to which conversion takes place. One always tries to find methods by which one can go back to that part of the body which provides important experiences in early childhood. The patient described is probably alcoholic because he tries to recapture the feeling of lightness of the body and to reproduce some of the sensations he felt when submerged by his mother. With the help of the concept of the body image one comes to deeper understanding of what is going on in conversion, especially in conversion à deux. One body image goes into the other body image. There is projection and introjection, the taking in of parts or of the whole of the body of the other person. The body image is not a static entity. One often overemphasizes the fact that when an identification has taken place it will last forever. Dr. Oberndorf's observations show clearly how the patients change. Sometimes the man identifies himself with his mother; sometimes he projects the picture of the mother into his wife, so that the identification is not static; it is dynamic and in continual change. The body image is constructed and reconstructed.

I now come to the topic of Dr. Jelliffe's fascinating paper. The construction and reconstruction of the body image leads to somatic disease, which is an attempt to reconstruct the body image. Something is pushed out of the body; something is taken in. In organic disease there is either wasting or swelling. These are

simple principles. One possesses the tendency to destroy parts of the body image. I doubt very much whether one ever has strivings to destroy the whole body. My conception in this respect is different from the freudian one; I believe that no one dies psychologically; I think that we die only by accident. I have found this to be true in seventy-five normal persons to whom Dr. Bromberg and I sent questionnaires. I have not found the freudian death wish in a dozen analyzed cases in which particular attention was given to this question. I am now studying the problem in the psychology of children; I have yet to find any human being who dies psychologically. Human beings are all living. They are all like the fire-bird, Phoenix, which is continually resurrected from its ashes. Man destroys his body image, but he builds it up again. One may call these self-destructive tendencies, but I should not like to call them death desires, for I believe in an everlasting reconstructive tendency of the psyche. I believe it is true that one has to destroy much in one's own body, and Dr. Jelliffe has justly pointed out that in studying the self destructive tendencies one may come to a deeper understanding of somatic disease and also of somatic health. In psychoanalysis at the present time one is astonished not so much when human beings are sick; one is more astonished when one finds them healthy, or at least almost healthy. Maybe the same question should be asked in somatic pathology—not why is a person sick, but why is he healthy. One may then come to a deeper insight into the strong tendencies to life. I doubt whether one will find real strivings for death.

DR. FRITZ WITTELS: One case in Dr. Jelliffe's paper particularly interested me. It concerned the girl who had a disease of the gallbladder and on whom no operation was performed. It was not Dr. Jelliffe's fault that exploration was not performed; incredibly enough, it was the fault of the conservative surgeon, who refused. After the patient was cured she fell into a state of depression that lasted for years. Dr. Jelliffe believes that it would have been better if an operation had been performed, because her tendency to self-punishment (i. e., her death instinct or self-destructive instinct) would have been thus satisfied and there would have been no need for the development of depression. I cannot entirely agree with Dr. Jelliffe. It is against my medical conscience to surrender to surgery a disease of the gallbladder or any other condition that can be cured without an operation. This is the more inadvisable in cases such as this in which the inflammation of the gallbladder might have been a symptom of hysterical conversion. Yet thousands of such cases are submitted to surgical treatment from time to time. From the psychoanalytic standpoint, however, symptoms of inflammation of the gallbladder or other physical symptoms of the hysterical conversion type unquestionably indicate the desire for punishment, and an operation gratifies that wish, temporarily releasing the patient from his ills. But the symptoms do not have that one meaning only. The power, importance and main interest of psychoanalysis consist in the discovery that symptoms have one or several meanings. They speak, but one cannot grasp the meaning of a disease of the gallbladder or of any other symptom of conversion unless it is analyzed. The symptom conveys a wish, but it is by no means only a wish for self-punishment.

As an example of the change of a physical symptom into a psychic symptom and of the occasional changing back of the psychic symptom into another physical symptom, I cite the case of a boy who consulted me because of a phobia. He was 17 years of age when it developed. Before the phobia appeared he had an ulcer of the stomach for years. An operation was not performed; the case was treated by medical means. Nothing brought relief until suddenly the symptoms of the ulcer disappeared. In their place a phobia developed which made life almost unbearable for the patient. Analysis brought to light the fact that the patient's father, whom he greatly loved and admired, had left his mother; the patient was the only son. His admiration for the father increased after the latter had displayed the courage and power needed to effect separation from the mother. The boy had only one objection to the father's departure; i. e., he should have taken him. The patient was left with the mother; the father remarried. An ulcer of the stomach developed in the boy, which I believe was a fantasy of pregnancy.

That symptom gave expression to his wish to be with the father; it said: "Why don't you take me? I am a boy; I am a girl also; I will give you a child; why don't you live with me? That second wife of yours should not become the mother of a child, because then everything would be lost to me, and I wish to live with you." The boy was sent by his mother to ask the father for money, and he suffered the humiliating experience of waiting in the hall until his father came out and said, "I have no money," and sent him away. He could not say to his father, "Marry me," because that is an impossible idea for the conscious ego. He continued to have the ulcer until the father's second wife gave birth to a child. After that the symptoms of the ulcer had no more value; they disappeared and gave way to a phobia. The unconscious meaning of the phobia was: There is now only one hope for me, that my mother should die, then father would take me. Of necessity this is a very superficial analysis. Had an operation been performed on the boy it might have helped him, it is true, inasmuch as he had the tendency to self-punishment; but that was only a part of the neurosis. There were other elements in the neurosis that could not be satisfied by an operation. The thing to do in cases of this kind is not to operate but to analyze.

I wish to comment on one of Dr. Meyer's statements, namely, that in cases of depression the reasons given by the patient for the depression are not the real ones. Dr. Meyer explained that it was clear to his patient why he was depressed: he was homosexual, and his friend had given him up. But that was not the real cause, and Dr. Meyer knew it, because he was well aware of the fact that people suffering from depression always screen the true reasons. Financial worries cause many people to commit suicide, and many are depressed in these times. But I believe that no case of melancholia or even of suicide is due to economic status alone. The victims state that their financial condition is responsible, but there are always other factors that one cannot so easily mention. One cannot say, "I am in love with my own son; I do not want him to marry." The superego does not permit it.

In Dr. Oberndorf's case the two people met, fell in love at first sight and married speedily thereafter. I believe that love at first sight is always pathologic. It suddenly appears because the other person has the quality or qualities that have been sought for and long repressed. Romeo and Juliet were pathologic. Their families hated each other. In Dr. Oberndorf's case it was clearly love at first sight; the couple met and after a week or so were engaged. Things like this happen again and again. That such cases are pathologic is important from the psychic standpoint. Parents come and say, full of pride, "Our children met and fell in love at first sight." One should say to them: "They should be analyzed, because marriage is not to be taken so lightly." When one observes the frequency of divorces one feels that a great deal of misery might have been avoided.

On the other hand, I do not wish to give the impression that love could ever be anything else but the result of former needs. If Freud had taught only one thing, that an element of mother-love exists in every love, he would have handed down a valuable theory.

Book Reviews

Mental Deficiency Due to Birth Injuries. By Edgar A. Doll, Ph.D., Winthrop M. Phelps, M.D., and Ruth Taylor Melcher, M.A., New York. Price, $4.50. Pp. 289. New York: The Macmillan Company, 1932.

On reading the title of this book one is led to expect a comprehensive treatise on the subject of the relationship of injuries sustained at birth to mental deficiency. It is disappointing to find, however, that the book is based on an analysis of only twelve cases, some of which are definitely doubtful. The preface indicates that the work has been confined as nearly as possible to cases of "(1) Mental deficiency more or less directly traceable to (2) cerebral injuries (3) apparently sustained at birth." Neurologic examinations are alleged to have been made for the most part by an orthopedist, which naturally opens the premises of the book to criticism. Some of the bad neurologic impressions made in the earlier chapters, however, are in a measure compensated for by more scientific statements on psychology in some of the succeeding chapters. It is assumed, a priori, that in all cases there were traumatisms at birth, with subsequent cerebral hemorrhage (probably of gross character) which resulted in paralyses, yet many of the case records would cast serious doubt on such an interpretation.

In the chapter on etiology the authors state that "there are two ways in which motor re-education might affect physical development: (1) By educating nerve centers to perform the natural duties of those destroyed and (2) by eliminating the bizarre cortical habit patterns which have been established and substituting in their stead more normal, simpler patterns of muscular activity." One can only marvel at the naiveté of such a statement. Only a psychologist could teach a neuron to perform a function which it was originally never intended to perform!

The cases studied are grouped into two classes. Group 1 contains seven cases, two of "extrapyramidal lesions characterized by athetosis"; two of "pyramidal lesions, characterized by spasticity"; one of a "diffuse lesion, pyramidal and extrapyramidal, characterized mainly by spasticity"; one of "probable brachial palsy, but associated with macrocephaly and speech defect," and one of excitability, with no evidence of motor lesion.

Group 2 is comprised of five cases, which are separated from those of group 1 because "elements in the family history make it difficult to say whether mental deficiency is due solely to the hemorrhage" even though the examiner expressed the opinion that the neurologic symptoms were due clearly to cerebral hemorrhage. Of this group, one case presented pyramidal lesions characterized by slight spasticity; three showed extrapyramidal lesions characterized by athetosis, and one presented an intention tremor and possible "neuropathic inheritance." The only evidence of pyramidal lesion in one case was slight hyperactivity of reflexes; the patient's mental level was between 9 and 12 years, and, in addition, there was considerable suggestion of congenital syphilis in the case. In another case birth was premature, and there was a positive Wassermann reaction. A third case is classed as "extrapyramidal," but showed in addition a bilateral Babinski sign and a mental level of 13 years (sic). In case 2, it is admitted that there was possibly other injury than that sustained at birth, and birth occurred two months prematurely; the outstanding features were intention tremor and an intelligence quotient of 87.

The fourth chapter is entitled "Birth and Developmental Histories," and on the whole is of some value from a statistical point of view. Well prepared tables summarize the facts nicely, but are based on the assumption that in each case a cerebral hemorrhage was caused at birth because of injury.

The fifth chapter, entitled "Mental Tests and Measurements," shows thorough work not only in devising methods for examining physically handicapped children

with pronounced motor defects, but also in the value of systematic efforts to arrive at a reasonable conclusion regarding mental ability. In this chapter of eighty-two pages the author (presumably the psychologist) is on familiar ground and handles the subject with clarity, precision and vision. It is concluded that "out of a battery of eight tests only two, namely the Binet and the Myers, gave results of much practical value for purposes of determining the intelligence level of birth-injured subjects."

Chapter 6, entitled "Mental Estimates," and chapter 7, entitled "Mental Growth," carry out the careful work of the preceding chapter and contain really good analytic material.

There is a chapter entitled "Physical Therapy," which offers nothing new, but reemphasizes the value of an attempt to train children who are handicapped in the mental as well as in the physical spheres. It seems to be shown, however, that improvement is possible only when the child possesses sufficient intelligence to cooperate properly, and that no progress can be made when the mental possibilities have not been fully reached, all of which is well known.

As an effort in psychologic interpretation of neurologically handicapped children the book is a valuable contribution, but it definitely fails to establish its main purpose—the relationship of mental defect to injuries sustained at birth.

Grundzüge der Psychoanalyse. By Leo Kaplan. Second edition. Price, 16 marks. Pp. 397. Baden-Baden: Merlin-Verlag, 1929.

The second edition of this book has its full justification in the face of the appearance of Freud's own lectures in 1917. Kaplan gives a specially careful account of repression and its explanations on ground of a dynamic conception of mental activity. Moreover, he aims to bring out the close relation of many factors in the process of repressions to the magic ways of thinking, and the relation of magic to narcissism. He outlines the relation between psychoanalysis and psychology and defends psychology against reflexology, in the face of the modern trend in the direction of quantity and technic, and the deeply rooted preference for objects that do not lie to a child in contrast to the lies of parents and of human utterances generally. The giving up of hypnosis and its replacement by overcoming resistance are attained by the encouragement of absolute freedom from blame in free association. Positive transference comes with the assumption of the impartial attitude of the analyst; it is narcissism that creates the split between knowledge and faith. In successive chapters, Kaplan discusses lapses and symptomatic acts; displacements and their dynamic foundations; condensation; repression in the unconscious (in a specially well elaborated and well documented presentation); the association experiment, the many modes of presentation by myth; the child and the primitive and the unconsciousness; dream, myth and neurosis; the transformation of affects and the primary motor impulse; hate and the sexual drive and its dynamics. The notes gathered in an appendix offer many instructive references to the literature and sources. Altogether the book is distinctly worth reading.

Grundzüge der Entwicklungsgeschichte des Menschen in vergleichender Darstellung. By Richard Weissenberg. Thirteenth edition. Price, 13.50 marks. Pp. 450. Leipzig: Georg Thieme, 1933.

The thirteenth edition differs little from the twelfth, which was published two years ago. The neurologic part is the same. The printing, however, and the illustrations are much better. It is an excellent presentation of not only the nervous system, but the whole body.